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19TH JUDICIAL DISTRICT COURT
FOR THE PARISH OF EAST BATON ROUGE
STATE OF LOUISIANA

----------x

ROBERT C. GILBOY,

Plaintiff,

- versus -

THE AMERICAN TOBACCO COMPANY, et al.,

Defendants.

----------x

Civil
Docket "I"
No. 314,002

September 20, 1995
10:00 o'clock a.m.

599 Lexington Avenue
New York, New York 10022

DEPOSITION of LAWRENCE GARFINKEL, a witness in the above entitled matter, pursuant to Notice of Deposition, before a Notary Public of the State of New York.

A P P E A R A N C E S :

GEORGE R. COVERT, ESQ.
Attorney for Plaintiff
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CHADBOURNE & PARKE LLP
Attorneys for The American Tobacco Company
30 Rockefeller Plaza
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BY: BRUCE G. SHEFFLER, ESQ., of Counsel

- and -

BY: DEBORAH A. BARNHART, ESQ., of Counsel

A P P E A R A N C E S (Continued) :

JONES, DAY, REAVIS & POGUE, ESQS.
Attorneys for Defendant, R.J. Reynolds Tobacco Co.
North Point
901 Lakeside Avenue
Cleveland, Ohio 44114

BY: THEODORE M. GROSSMAN, ESQ., of Counsel

- and -

BY: MARK A. BELASIC, ESQ., of Counsel

L A W R E N C E G A R F I N K E L, called as a witness, having been first duly sworn by the Notary Public, was examined and testified as follows:

EXAMINATION BY MR. GROSSMAN:

Q What is your full name?

A Lawrence Garfinkel.

Q Mr. Garfinkel, we introduced ourselves earlier, but for the record I am Ted Grossman.

I am a lawyer from the law firm in Cleveland and I represent the R.J. Reynolds Tobacco Company in the Gilboy case.

Before we get on with questioning, since your testimony has been noticed by the Plaintiffs in a number of cases, I just want to clarify that this first deposition is exclusively for the Gilboy case.

MR. COVERT: That's correct.

MR. GROSSMAN: Although, obviously, any sworn testimony in one case can be used in another for impeachment purposes.

Q We have an agreement in this case as opposed to the Marks case, which we will come to later, that your testimony, Mr. Garfinkel, is limited to the relationship between smoking and peripheral adenocarcinomas and scar cancers.

Have you discussed that with Mr. Covert?

A Yes, I did.

MR. SHEFFLER: Before we go any further, I just want to note that American Tobacco Company objects to the addition of Mr. Garfinkel as a rebuttal witness in the Marks case.

Q Mr. Garfinkel, you understand in the context of the Gilboy case you will not be commenting on the risks of cigarette smoking to society or the number of deaths that could be attributable to cigarette smoking?

A I will only reply if you ask me a question about that.

Q But that's not within the context of the testimony that you are prepared to offer, is that correct?

A Okay.

Q All right, I will be asking you a

series of questions today. If you don't hear me, will you let me know?

A Yes.

Q If you don't understand the question, will you let me know?

A Yes.

Q I know from your forty years of writing that you have been involved in epidemiology and some other subjects in an intensive manner.

I may use some terms inappropriately.

A All right.

Q If I do, will you let me know that?

A I will correct you if you do.

Q Some of my questions may call for a discoursive answer and others of my questions may fairly call for an answer of yes or no.

Will you listen carefully to the questions?

A All right.

Q If they call for a discoursive answer, will you give that type?

A Will I what?

Q Give a discoursive answer if the question fairly calls for one?

A If the question calls for one, I will try.

Q And if the question fairly calls for an answer of yes or no, will you answer that way?

A Sometimes when a question is -- if you are asked to answer a question yes or no, it doesn't give you the full flavor of what my opinion is.

Q If it fairly calls for an answer of yes or no, will you answer that way?

A Yes, I will.

MR. COVERT: I would like to, I think, only in fairness to Mr. Garfinkel, explain that he always has a right to explain his answers, even yes or no.

Q When were you first contacted by Mr. Covert?

A A month ago, two months ago, something on that order. I probably have the letter; in that neighborhood.

Mr. Covert has the formal --

Q You can take your time.

A Here is a letter of August 18th that tells me about depositions and that was probably two weeks or so after his first contact with me.

Q How did Mr. Covert first contact you?

A By telephone.

Q So, it's your recollection that you were first contacted when?

A It was probably sometime in August, early August.

Q Of this year?

A Yes.

Q Could you give me a copy of the letter that Mr. Covert sent to you?

THE WITNESS: Do you have a copy of this?

A I think that's the only letter I have gotten from you. This is my entire file.

MR. COVERT: Okay.

Q May I see that?

A Here (handing).

MR. GROSSMAN: Jerry, at a break I will have this copied and we will be able to make an Exhibit 62.

Q Mr. Garfinkel, I see you have brought a file with you.

Is that your file in the Gilboy case?

A Well, it's all the papers that pertain

to it, and the correspondence, yes.

Q Do you mind if we examine the file?

MR. COVERT: It's okay.

Q Mr. Garfinkel, I see that you have the Federal Judicial Center's reference manual on scientific evidence?

A Yes.

Q Was that provided to you by a lawyer, or is that something that you looked at?

A Mr. Covert sent it to me.

Q You also have a copy of your article written with Oscar Auerbach and Verda Parks in 1979 for Cancer Magazine called "Scar Cancer of the Lung"?

A Right.

Q And your 1991 article with Dr. Auerbach on the changing pattern of lung carcinoma?

A Right.

Q An article from The Lancet, it appears, in 1981 by Dr. Auerbach on the pathogenesis of lung cancer?

A Yes, that's an article that Mr. Covert had in his possession that I was unaware of. Let me see that paper.

Q Sure (handing).

A Yes, this didn't appear in the Lancet, it appeared in some journal called Comprehensive Therapy.

Q So this was provided to you by Mr. Covert?

A By Mr. Covert. I thought I had every article Dr. Auerbach wrote, but apparently I didn't.

Q There are things marked here that appear could have been marked in pen or pencil?

A That is a copy of a paper that Mr. Covert provided me and that's yellow highlights that he put in the paper.

Q The highlighting is not yours?

A No.

MR. COVERT: That was taken from Dr. Martin's deposition in Gilboy.

Q There is another article here by Falk, Pickel, Phantam, Greenberg, Jacobs, Correa and Fermany on the Epidemiology of Bronchioloalveolar Carcinoma?

A That was also provided to me by Mr. Covert.

Q Did he tell you why he was providing

you with that?

A He mentioned that there was an article which found that cigarette smoking was related to bronchioloalveolar carcinoma and I had been unaware of that article.

So he sent it to me -- he faxed it to me actually.

MR. COVERT: That would be an Arabie article.

Q That is for Arabie?

A Correct.

Q There is a work sheet here?

A That was just notes for myself.

Q Well, you don't have to worry about my trying to read them, just as I don't worry about people trying to read my notes.

A They have trouble reading doctors' handwriting, but they also have trouble reading epidemiologists' handwriting.

Q There is an article here by Barsky, Cameron and others, " Rising Incidence of Bronchioloalveolar Lung Carcinoma and Its Unique Clinicopathological Features."

Is that from your file?

A That's from my file.

Q Dr. Auerbach, you, along with Hammond, Kirman and others had an article on the Histologic Changes in Bronchial Tubes of Cigarette Smoking Dogs?

A Right.

Q That's from your files?

A Yes.

Q A 1967 JAMA, again from your files?

A Yes, that's from my files.

Q There is a letter addressed "To Whom It May Concern" from Sanford Barsky.

A That was provided to me by Mr. Covert.

Q That's for the --

MR. COVERT: That's for Arabie.

MR. GROSSMAN: That's for the Arabie case.

Q This is another letter dated August 28th from Mr. Covert. We'll have that copied and we can ask questions about it when we make it an exhibit.

Doctor, I note two pages of notes in here with various numbers. Could you tell me what those are?

A This is an analysis based on the fifty-five cases in the changing pattern of lung cancer paper, in which I looked at the percentages in table 4, I think, of the article, that estimated the number of cases, which is not shown in there.

Q Is that something that you prepared recently?

A Yes, this was since I was first contacted.

Q So since August?

A These are other worksheets which look at -- I did -- let's start over again.

Q Okay.

A There is a letter there which express how -- my estimate of whether or not BAC is related to smoking.

I made several assumptions in that letter and these are some of the notes that I made to myself in preparing that article.

The top here is all but the BAC cases, and it shows a 22 to 1 relationship with smoking.

The second one here is making some assumptions on the smoking, distribution on extreme conditions, saying there is -- most of the people in

our -- in our fifty-five lung cancers were still smoking, although there was some -- there were a number of ex-smokers, and it's another -- it's another attempt to estimate the relative rate -- risk of smoking under such extreme smoking distribution assumptions.

MR. COVERT: Since we are having duplications, cross-references, I am just going to down at the bottom put a BAC.

A This has to do with BAC.

MR. COVERT: So that will be identifiable that way.

MR. GROSSMAN: We can make copies of these and ask questions about them later.

I just wanted to identify what was on.

MR. COVERT: This one will be pertinent to Arabie, is that correct?

THE WITNESS: Yes.

Q Doctor, just for clarification, there is a letter here dated September 1, 1995 from you to Mr. Covert?

A That's what I referred to just before.

Q That's the letter about BAC?

A Yes.

Q Why don't we hold that out as well.

A There is a table that accompanies it.

Q Here is the table (handing).

A This again refers to the other case.

MR. COVERT: We will write it BAC2, Bruce.

Q You were provided with some pages -- let me start this again.

You have xerox copies of Pages 20 through 34 of what appears to be a deposition of Dr. Martin.

A Right.

Q Which I believe is the deposition from the Gilboy case, because I was present and Mr. Shev was present.

Were you provided with the rest of that deposition?

A No.

Q You were provided only with Pages 20 through 34?

A That's what you sent me.

Q What Mr. Covert sent you?

A Yes.

Q Were you provided with any other

depositions in the Gilboy case?

A No.

Q Were you provided with any summary of testimony in the Gilboy case?

A There is a letter there from a doctor -- I forgot his name, he's a radiologist -- and there is one from Dr. Barsky.

Q Dr. Barsky is in the Arabie case?

A Yes.

MR. COVERT: He's a radiologist in the Arabie case.

Q So the only deposition material or opinion material that you were provided in the Gilboy case was Pages 20 through 34 of the deposition of Dr. Martin?

A Yes.

Q I will give you back the rest of your file (handing).

Were you provided with any letters by Mr. Covert, other than the letters that appear in your file?

A Excuse me, before I answer the question, there is one other sheet of paper which is pertinent to what we are talking about, and that is

an estimate of the relative risk of smokers who die of adenocarcinoma, peripheral adenocarcinoma and all adenocarcinoma.

Q We will mark that in a moment.

A This I just did very quickly yesterday, because I was unaware that one of the cases was peripheral adenocarcinoma and it's not properly labeled.

Q We can copy that at a break and then ask questions about it later.

Q Did Mr. Covert provide you with any other materials of any kind or any other letters for the Gilboy case?

A I don't believe so; everything he provided me is in these files.

Q Now, do you know anything about Mr. Gilboy or his illness, other than what was contained in the materials that you were provided by Mr. Covert?

A I know he presumably died of a peripheral adenocarcinoma. That's all I know about it.

Q Were you ever provided with his autopsy report?

A No.

THE WITNESS: Do you have it?

MR. COVERT: No, not with me.

MR. GROSSMAN: Let's mark this as Garfinkel Exhibit 1.

(The above described document was marked Garfinkel Exhibit 1 for identification, as of this date.)

Q Mr. Garfinkel, let me hand you what has been marked for identification purposes as Garfinkel Exhibit 1.

I am handing you this just so we don't operate in the dark here, since you are under the impression, apparently from your discussions with Mr. Covert, that Mr. Gilboy had died of adenocarcinoma.

This Exhibit 1 is the autopsy of Mr. Gilboy and it shows the cause of death as pulmonary embolism and bronchial pneumonia.

If you look at the second to last page, 6, of this autopsy.

A Page what?

Q Page 6, second paragraph, it says, "The autopsy failed to show any neoplasm. Patient

probably died of respiratory failure due to marked pulmonary fibrosis, pulmonary emboli and bronchopneumonia.

"The diagnosis of malignancy in this patient cannot be substantiated on the basis of this autopsy procedure."

A May I make a comment on this?

Q Yes.

A Paragraph 2 of Page 2 says, "64 year old white male, reportedly heavy smoker, who has a right upper lobe lesion for a number of years, diagnosed as adenocarcinoma, which later clinically and surgically metastasized to the brain, for which he underwent a craniotomy procedure."

Having had an adenocarcinoma and having had it removed, with no evidence of residual disease, results in not seeing any cancer on autopsy.

Q Mr. Garfinkel, what I was showing this to you for was to remove any impression that you may have had that Mr. Gilboy had cancer at the time of his death.

I think it's been stipulated between the parties that he had no cancer at the time he

died.

In 1986 --

MR. COVERT: Well, I don't think it's been stipulated. I think there is no discernible cancer at the time of his death.

MR. GROSSMAN: In answers to interrogatories, Gerry.

MR. COVERT: Whatever, that will speak for itself.

MR. GROSSMAN: In any event, Mr. Gilboy was diagnosed with cancer in 1986, he received treatment for it.

By 1993, at the time of his death, he had no cancer.

All right, let's move on.

Q How many times have you spoken with Mr. Covert or others from his office?

A I can't remember; it may have been three or four times.

Q Have you spoken with Sean Fagan of his office?

A No.

Q Or James Piker?

A No.

Q Only with Mr. Covert?

A I spoke to his secretary.

Q Now, when Mr. Covert spoke to you in August of this year, did he tell you that you had previously been listed as a witness in any of these cases?

A I have never appeared as a witness in a lung cancer smoking case.

I once gave a deposition in lieu of the unavailability of my associate, Herb Seidman, in an asbestos case. I wasn't directly involved but I knew enough about the case that they asked me to give a deposition.

Q That was involving the subpoenaed deposition for Mount Sinai records?

A Yes -- well, I don't know what it was. it may have been involved in that; I don't recall what it was.

All I testified to was the history of the studies that the Cancer Society participated in in conjunction with the Mount Sinai people.

Q Have you ever been deposed in any other kind of case?

A No.

Q So today, apart from the deposition involving the Mount Sinai and American Cancer Society documents, today is the first deposition you have ever been involved in?

A Right.

Q Have you ever testified at trial?

A No.

Q Have you reviewed all the materials that will be necessary for you to form all the opinions that you intend to render at your trial testimony tomorrow?

A All that I am aware of.

Q If at any time in the future you review other materials that make you believe it necessary or appropriate that you revise your opinion, will you inform Mr. Covert so that he can provide us with those materials and with that additional information?

A Or if he -- yes, I will, or if he provides me with other articles of which I am unaware, we will certainly let you know.

Q Just to clarify a previous point, when Mr. Covert contacted you in August of this year, a month ago or a month and a half ago, did he tell you

that he had previously identified you as a witness in any cases?

A I don't recall. I just don't remember.

Q Is it fair to say that before August of this year you certainly didn't know that you had been identified in any cases as a potential witness?

MR. COVERT: Ted, I think that Mr. Garfinkel's report may be incomplete, we had a discussion in March of 1994.

THE WITNESS: Did we?

MR. COVERT: Yes.

THE WITNESS: I just don't remember this. Is it possible I never received it?

Did I ever talk to you about this?

MR. COVERT: Yes.

THE WITNESS: I did call you; I really have no memory of it at all.

A Just for the record, I do recall, many years ago, it may have been ten years ago, some attorney who had contacted Dr. Auerbach -- who had contacted Dr. Auerbach about testifying -- Dr. Auerbach has testified a lot -- came up to see me and asked me if I would testify in a trial.

My policy then was when I was a staff member of the Cancer Society, I retired in 1990, that I would not testify in any legal cases.

But that's the only other contact I remember. I simply don't remember that at all.

Q That's all right, we will make a copy of it and we will put it into the record later.

Your report indicates that your compensation is $250 for the report and $500 for depositions.

Just for the record, how did you set your rates, in talking to others who had been deposed?

A No, I didn't talk to anybody who had been deposed.

My son is an attorney and I spoke to him about it. He thought it was kind of low; I just pulled a figure out of a hat.

Q Since it doesn't give time frames, when you have $500 for the deposition, what period of time are you referring to?

A Well, I assumed that a deposition would take about an hour or so.

I have no idea that I would be asked

to appear at this hearing and I will send you a bill for this, too.

Q That's fine, but the rate that you are referring to was, I think, to be a deposition that would last only one day?

A It would last one day, I thought, maybe a couple of hours, and then to review the papers -- I thought it was mostly the changing pattern of cancer, maybe one or two others.

I thought that would just take me a short period of time to refresh my memory about what we said in that paper.

Q Just so I am clear about this, the amounts that you are talking about are amounts for the deposition itself or for the deposition and preparation?

A Deposition and preparation.

Q What, if you could break it down, just to itemize it, what portion would be for preparation and what portion would be for the deposition?

A Well I said in the -- I think I said in the letter $250 for preparation and $500 for the deposition.

Q $250, when you said for the report,

that's the preparation time you are referring to?

A Yes.

Q Okay.

A When I set that I didn't realize at that time there would be much more than that, reading other articles and so forth, but that's all right.

Q Well, based upon previous rulings in this case, the preparation time is something that Mr. Covert owes you and the deposition time was something that we owe you, and that's why I am trying to clarify that.

If you pass along your bill for the deposition in time to Mr. Covert, he will send it to us, then we can forward the fee to you.

MR. COVERT: That's fine.

A How about for this time we are spending today?

Q That's what I am talking about.

A This is deposition time?

Q This is deposition time, then tomorrow is a separate deposition time. I assume when you sent that to Mr. Covert, you assumed it would all be in one day and obviously this is two days.

A Yes. Well --

MR. SHEFFLER: The deposition time tomorrow, Mr. Garfinkel, you will send to Mr. Covert.

THE WITNESS: I sent that to him already.

Q I assume you are charging Mr. Covert the same rates that you are charging the Defendants?

A Same rate what?

Q Same rate that you are charging the Defendants?

A Yes.

MR. SHEFFLER: Let's have this marked as Exhibit 2.

MR. COVERT: That the CV?

MR. GROSSMAN: Yes.

(The above described document was marked Garfinkel Exhibit 2 for identification, as of this date.)

Q Mr. Garfinkel, let me hand you what has been marked for identification purposes as Garfinkel Exhibit 2.

Mr. Garfinkel, is that a current CV for you?

A Yes, it is.

Q This indicates that you received a Master's in sociology in 1949 at Columbia?

A That's right.

Q Have you had any formal education leading to a degree since then?

A Since 1949, no.

Q Beginning in 1949, where did you first work, that's not clear from here?

A I started work in the American Cancer Society in 1947.

Q You worked there until 1990, when you retired?

A I worked there until 1990 when I retired as Vice President of Department of Epidemiology and Statistics and since 1990, the end of the third paragraph, I have been senior consultant to the Cancer Society in epidemiology statistics.

Q When you arrived at the Cancer Society in 1947, was there a Department of Epidemiology and Statistics?

A It was called the Department of Statistical Research at that time.

Q When did the name epidemiology first arise?

A I can't remember, probably in 1970 or '75, around there. Let me just -- I could check it by looking at one of these older papers.

In 1967 it was still known as the Department of -- the Statistical Research Section, it was called. Let's see if I have any papers here.

In 1979 it was the Department of Epidemiology and Statistics.

So I guess about 1975 is probably best.

Q Were there academic disciplines of epidemiology or biostatistics in the 1940's, to your knowledge?

A Very, very few. As a matter of fact, we in the early days, in the '40s and '50s, Dr. Hammond and I, we we didn't call ourselves epidemiologists, we called ourselves biostatisticians.

The term epidemiology was around, but it evolved as a discipline, graduate school was much later.

Q So, your formal training was in

statistics when you were going for your Master's in sociology?

A Right.

Q And you applied that to medical issues at work?

A Yes.

Q Now, your CV also lists you as having worked in the Department of Removable Prosthodontics?

A Right.

Q At the NYU School of Dentistry.

What was your work in that?

A Well, about one day a week, one morning a week, I would meet with students at the New York University School of Dentistry and advise them on their Master's thesis.

These were graduate dentists going for a Master's Degree in prosthodontics, and part of their responsibility was to write a research paper.

During that time I also taught a class in statistics to graduate students, not only in the Department of Removable Prosthodontics, but also the other graduate departments of the New York University School of Dentistry.

Q You also list here work on the Advisory Committee of Diet and Nutrition Cancer Program?

A Right.

Q And the clearing house on environmental carcinogens, a member of the advisory group of the SEER Program at the NCI?

A Right.

Q What was the SEER Program?

A The -- SEERS, a program for surveillance epidemiology and end results.

It's a program set up by the National Cancer Institute in 1973 which tries to obtain all the cancer cases in areas that comprise about one-tenth the population of the United States.

It's the best source we have for incidence data on cancer. It started in 1973 and it's ongoing ever since.

Q When you say it's the best source that we have --

A For incidence data, yes.

Q (Continuing) -- for incidence data, are you referring exclusively to incidence, or are you referring to mortality as well?

A Well they report mortality, too, on their population, but mortality data is generally taken from official statistics of the National Center For Health Statistics.

But the incidence data is not available from that organization and we have to rely on this sample of the total country for good incidence data.

Q So the SEER Program is a sampling program to determine incidence of various forms of cancer?

A Right.

Q It's based on reporting by doctors?

A It's reporting in hospitals. They don't get reports from doctors' offices.

They have every hospital in certain proscribed areas involved. There are five states and five other areas that are combinations of several counties.

Q As part of the SEER Program there is no effort made to doublecheck the accuracy of the diagnoses, is there?

A They get pathological reports. I think one of the last reports said at least

ninety-six percent, I think, maybe ninety-eight percent, of every cancer is microscopically confirmed.

Q By the SEER Program?

A By the hospitals in the SEER Program.

Q Yes, but when you say biologically confirmed, there is one pathologist who reads the slide?

A Oh, no, these are the histological reports from the hospitals from which the data are gathered.

Q Let me rephrase the question.

You have written yourself on inter-observer variability among pathologists, is that correct?

A I don't know if I wrote a -- could you refresh my memory which paper you are talking about? I know there have been a number of papers on that subject; I don't recall writing one myself -- oh, I know one.

Q Mr. Garfinkel, it's fair to say you have been a co-author on a number of articles, isn't it, as well as an individual author, is that correct?

A Oh, yes, I have been a co-author.

Q When you are a co-author of an article, do you review it in full prior to publication?

A Most of the time, but not necessarily all the time.

Q Is there any time you can recall that you did not review an article fully prior to publication when the article carried your name?

A I can't recall one right now.

Q Is it fair to say, doctor, that if there were anything in an article bearing your name with which you disagreed, you would have the article changed?

A It depends. Sometimes authors of an article may have different opinions on the interpretation of the data they do and they discuss it and decide what is important and what isn't important.

MR. GROSSMAN: Let's mark this as Garfinkel Exhibit 3.

(The above described document was marked Garfinkel Exhibit 3 for identification, as of this date.)

Q Mr. Garfinkel, let me hand you what has been marked for identification purposes as Garfinkel Exhibit 3, which is an article from CA-A Cancer Journal For Children which is entitled "Classification Of Data And Research."

You are listed as the only author of this?

A Right.

Q It's from January/February, 1979.

If I could direct your attention to the second page of that, which is actually page number 3 of the journal.

If you look in the left-hand column, you note "Investigators without full knowledge of definitions used should always be cautious in comparing results of different studies appearing in the literature.

"Although" --

A I'm sorry, I don't follow where are you on the page.

Q Right up here (indicating).

A Investigators.

Q "Investigators without full knowledge of definitions used should always be cautious in

comparing results of different studies appearing in the literature.

"Although different investigators may put the same name to a diagnosis or a procedure, it's not always clear they are referring to the same entity.

"For example, a study comparing the histologic type of lung cancer of the same slides read at different periods of time showed a thirty-seven percent discrepancy."

A Let's see what the reference was. Okay.

Q Then you say, "Definitions and concepts do change over time. There also may be considerable variability among pathologists reading the same slides."

There is, in fact, significant inter-observer variability that's been noted in the literature on pathologists, is that correct?

A Right.

Q And therefore -- and also, not only has there been inter-observer variability that's been noted in the literature, but there has even been variability when the same pathologist has read

the slide, same slide, more than once in studies, is that correct?

A That seems to be correct, in reference 4.

Q Now, in the SEER Program, there is no effort to doublecheck the hospital's diagnosis of cancer or cell type, is that correct?

A Only what the hospital itself does. Many hospitals may have -- certainly when there is difficult cases, it would have more than one person in the pathology department reading it.

So they have their own set of references.

Q The hospital does whatever it does normally.

A The SEER Program does not have one pathologist who reviews all the slides, cases.

Q It doesn't have any pathologist who does that, is that correct?

A They may do it for special purposes. If they are doing a specific study of one particular pathological type the pathologist at the SEER headquarters may ask for those slides from the hospital, to review them himself.

Q As a matter of general course, the SEER Program does not ask for slides from the hospitals?

A No.

Q As a matter of general course, the SEER Program relies upon the hospital's diagnosis in compiling its numbers?

A Right. You know, I should point out that the hospitals itself do not supply the SEER Program with the data.

There is generally one coordinating agency in each of these ten areas; it may be the state cancer registry or it may be a university or something, that collects all the data from the hospitals.

Now, some of them may -- cross that out.

The SEER Program has a very rigid program of checking the data that they receive for accuracy. They make all of the cooperating, coordinating agencies review the data very carefully.

Now, under that procedure -- whether that procedure involves having a pathologist look

over the slides at the coordinating center, I don't know; some of them may, some may not.

Q But you don't know if they do or not?

A I don't know.

Q Okay, Mr. Garfinkel.

We have established that you had a Master's in sociology, in which you majored in statistics, and that you have been working on statistical biological matters since 1947.

Do you continue to work on smoking and health research?

A I am currently involved -- I am almost finished now -- as an editor of a journal -- of a monograph which will be published by the National Cancer Institute, in which all of the known cohort studies, the two Cancer Society studies, the women's health study in Boston, the dog study, the Kaiser Permamente study, will all be updated with new data.

That publication should be out very recently.

My role in it -- I am a co-author on one of the papers -- but my role was as an editor to read it over and make comments to the author on what I think should be emphasized or not emphasized.

So that is my only involvement recently, I think, with smoking and health issues.

Q Are you still involved in other statistical issues?

A I still publish some papers. Since my retirement in 1990, I estimate I may have twenty papers published, but many of them are editorials, or rehashing of data.

There is one paper which is going to be published next month in Metropolitan Insurance Company statistical bulletin on the probabilities of developing and dying of cancer, but it's not original data, it's something I have taken from another publication.

Q You continue to publish from time to time in Cancer CA?

A Yes, two different publications. CA is -- it's a small Reader's Digest sized journal and the other one is the Journal of Cancer, which is a full sized medical journal.

Both are American Cancer Society publications.

Q Doctor, you have never received -- you have never received formal medical training?

A No, I haven't.

Q You have no expertise in diagnosing cancer?

A No, I haven't.

Q You have no expertise in determining whether cancer is primary or metastatic?

A Not clinically. I have some expertise in coding death certificates to determine which is the primary and which is the metastatic site.

Q That is to say, you review death certificates that have been written by doctors to determine what you understand the doctor to mean?

A Right. There are certain rules that one goes by and, of course, in the American Cancer Society studies, we received in the first study 70,000 death certificates, and it was my job to make the rules of coding according to certain standards, but adapted to our own purposes, and train people to do this.

Q Let me see if I understand you fully. As part of the American Cancer Society studys, you base your statistical compilations on material obtained from death certificates?

A Right.

Q You set up protocals for reviewing the death certificates to determine whether the death certificates indicated a primary or metastatic carcinoma?

A Right. The National Center For Health Statistics has certain types of rules for doing this.

We used those rules, but adapted them in some ways to fit our needs, and sometimes we coded, for example, the underlying cause, the second cause or the third cause.

The published data from the National Center For Health Statistics usually publishes only the underlying cause.

So in some ways we adapted it to fit our needs.

Q Well, let me hand you once again what has been marked for identification purposes as Exhibit number 1, which is a copy of the death certficate of Robert Gilboy.

For coding purposes at the American Cancer Society, what would you -- that's the autopsy protocol of Mr. Gilboy?

A You see, if this doctor filled out the

death certficate correctly, he would have -- I wish I had a death certficate, the death certficate says 1A, underlying cause, secondary cause, third cause, and then it says "other significant conditions."

In this case he would say the immediate cause of death was emphysema, he might say emphysema and atelectasis.

He would say probably bronchopneumonia and he may pick one of these as being a third cause.

This is not necessarily in the order in which he thinks it killed the person, they are just eight conditions that were present.

Then in the other significant conditions, he would probably put, or he should put, I would say, adenocarcinoma of the lung removed in 1987, or whatever it was.

Now, when we code this cause of death, depending on how he wrote it -- of course, we can't tell -- if we had the autopsy -- if I had this autopsy, I would probably not code this person as the underlying cause being cancer, because the doctor specifically says it wasn't.

In some other cases, depending how

close to death it was, these are rules that we've evolved over the years in consultation with pathologists, consultants, we might very well code lung cancer as the underlying cause.

Q So, if Mr. Gilboy's doctor had said that cancer could not be confirmed --

A Then we couldn't put it as the underlying cause, we would probably put cancer as a second or third condition, in our coding scheme.

Q In your coding scheme?

A Right.

Q If the doctor said that -- if the doctor noted that there had been a previous diagnosis of cancer, but did not note any finding regarding cancer in the death certficate, what would you then code it as?

A If we don't have it, we couldn't code it. We can't guess what it would be, obviously.

If, on the other hand, Mr. Gilboy was operated on in March of 1993 and at the time of the autopsy there was no evidence left of the cancer, and depending on how he wrote it on the death certificate, where that was -- if if he wrote it as a second cause, let's say, immediate cause

bronchopneumonia, lung cancer operated on three months ago -- we would code it as the underlying cause of death.

Q Let me turn your attention to Page 3, see where it says "head"?

A Yes.

Q The last sentence says, "The formerly black colored area of the sub-stantianigra appears light pink in color."

Do you see that?

A Yes.

Q If that had shown up on an autopsy report or death certficate, would you have coded that as the cause of death?

A That would not appear on the death certificate.

I looked at ~~tens of~~ thousands of death certificates; I have never seen such minute detail coded.

I could say that it's almost one hundred percent certain you would not see that on the death certificate.

Q So if depigmentation of the sub-stantianigra were noted in medical records

provided to you as part of an epidemiological survey, would you disregard that information?

A I would have no way of coding it. There is no rubric that covers that.

Q And having no rubric that covers that, you wouldn't code it, and because it wasn't coded, it would not enter into your system?

A It wouldn't enter into our system, right.

Q So if a medical finding -- just for clarification, if a medical finding were made by a medical doctor that an individual had depigmentation of the sub-stantianigra of the brain, the American Cancer Society data would not include that finding because there isn't a rubric to put it under?

A Certainly -- I mean, there's a lot of this in this -- if you look at Page 3, under "thorax," four lines from the bottom of that paragraph, "The parenchyma varies from tan brown to black in color and from spongy to meaty consistency."

That isn't coded. An autopsy is full of such descriptive terms, which is not a disease category.

Q Upon receiving this autopsy report, which notes on the first page "aspiration of stomach content and multiple pulmonary emboli," and which also lists "depigmentation of the sub-stantianigra," would it be called to your attention that the individual involved may have Parkinson's disease?

A Unless the doctor specifically says Parkinson's disease, we would not have that information, we wouldn't code it that way.

Q And you certainly wouldn't be in a position to diagnose Parkinson's disease on the basis of that information?

A Remember, I am not doing it, I am giving instructions to other people to do it.

Q Yes.

A But unless there is a disease category, you cannot code it.

When I say a disease category, I am referring to the international classification of diseases, the current revision.

Q Now, Mr. Garfinkel, you are not a pathologist, so you have no expertise personally in determining cell type of a particular lung cancer, is that correct?

A That's correct.

Q Your training in reviewing medical records is limited to trying to determine what the doctors have diagnosed rather than making diagnoses yourself?

A I don't make diagnoses myself. It's not just limited to coding of death certificates.

In working with Dr. Auerbach, the general format was that Dr. Auerbach would explain what he was looking for in a particular slide and I would decide a code, so that he could record what he had seen in numerical form.

Q But he's the one who lmade the diagnosis?

A But he's the one who makes the diagnosis.

Q Now, Dr. Auerbach and you wrote a number of articles together?

A Yes, we did.

Q Many of them were based upon autopsies. He performed those autopsies?

A As the head of the department, he didn't do the autopsies himself, but he reviewed the finding of his associates.

Q You didn't participate in those autopsies?

A No.

Q You didn't participate in the review of the slides?

A No.

Q Obviously, you cannot provide medical testimony regarding Mr. Gilboy, is that correct?

A I can't provide clinical data on his condition, no.

Q And any testimony that you will be giving is based upon statistics rather than upon medical expertise?

A If by statistics you include the whole field of nosology and classification of disease, then it's right.

Q What was the first word?

A Nosology.

Q What is nosology?

A Just what I said, it's the classification of diseases into a specific codes, and people who do it are called nosologists.

Q So, the testimony that you will be giving in this case is based upon your statistical

background and upon your work in nosology?

A Yes.

Q I assume you had never met Mr. Gilboy?

A No, I haven't.

Q Have you ever met his family or any of his friends or anyone who knew him?

A No, I haven't.

Q You have never read any depositions in this case apart from those sixteen pages or so of Dr. Martin's deposition?

A Yes; that's correct.

Q You have no information, apart from what has been provided to you, about the risk factors that Mr. Gilboy may have had for lung cancer, is that correct?

A No, I don't have any information on his background.

Q You don't know, for example, whether he was a uranium miner?

A I just see -- no, I don't. I just see he's reportedly a heavy smoker.

Q Obviously, if he were a uranium miner, that would affect his risk of lung cancer?

A It certainly should have been noted if

the doctor knew about it.

Q I am not saying that he was a uranium miner, but I am just saying if he were, that would be an example of something that would increase the risk of lung cancer and it would be something that you as an epidemiologist would want to know about?

A Certainly.

Q Similarly, if he had an extensive family history of lung cancer, that, too, would be something that you as an epidemiologist would be interested in?

A It hasn't really been established that a history of lung cancer predisposes somebody to develop lung cancer himself.

There are some reports that indicate it may be true; there are others that indicate it may not be true.

Q We can address those in greater detail later.

Just to sum up areas that you are not expressing an expertise in, you are not an expert in oncology?

A It depends on your definition of oncology.

Certainly, transient cancer, the statistics of cancer, mortality, morbidity, are all part of oncology and I am an expert in those.

Q When you say transient, you are talking about statistical trends?

A Statistical trends.

Q In terms of treatment of individuals?

A In terms of treatment of individuals, I am not an expert.

Q And the same would be true of radiology?

A That's true.

Q Neurology?

A Right.

Q Toxicology?

A Right.

Q Chemistry?

A No.

Q You have no expertise?

A No expertise.

Q Molecular biology, no expertise?

A No.

Q No expertise in psychology or psychiatry?

A No.

Q Or substance abuse?

A No.

Q Or pharmacology?

A No.

Q No expertise in cigarette design?

A Cigarette design. I do have some experience in analyzing mortality data related to whether or not cigarettes have filter tips or not, or the amount of tar and nicotine in the cigarettes.

Q You have found in -- you have reported in the literature that individuals who smoke filter cigarettes have a lower incidence of lung cancer than those who smoke similar amounts of regular cigarettes?

A The amount of tar and nicotine in cigarettes seems to definitely be related to the lung cancer rates, right.

Q The extent of your expertise in cigarette design is in the relationship between the tar delivery of cigarettes and lung cancer statistics?

A That's correct.

Q But you have no other expertise in

cigarette design?

A Explain what you are talking about.

Q You would not claim an expertise in determining how to make a cigarette that would deliver different tar or nicotine concentrations?

A If you are talking about the composition of the leaf or the manufacturing process, no, I don't have any expertise in that, no.

Q And you have no expertise in warning or communication theories, is that correct?

A In what?

Q Warnings theories and communications theories.

There are people who teach courses in the drafting of warnings and of communications generally with the public.

That's not an area of your expertise?

A If someone in the Cancer Society was going to get out a statement about harmful effects of cigarettes and guidelines for the department, or something of that sort, they probably would have showed it to me before.

Q They would show it to you and you might have an opinion?

A I may have an opinion on it, but not in designing the statement itself.

Q You wouldn't claim an expertise in how the public would react to that?

A No.

Q You don't claim an expertise in cigarette advertising?

A No.

Q And, obviously, you would defer to the conclusions of experts in all of those fields?

A Yes, I would.

Q Just to go through a couple of other things, then we can take a break to copy the materials that you have provided.

You are familiar with Ernst Wynder?

A Yes, I am.

Q He's an expert in the epidemiology of smoking related diseases?

A He is.

Q You are obviously familiar with Oscar Auerbach, having written with him?

A Yes.

Q You view him, as well, as an expert in the epidemiology of smoking related diseases?

A Yes. I'm sorry, I didn't hear the last thing. Is Oscar Auerbach an expert in the epidemiology --

Q Of smoking related diseases.

A No.

Q He's an expert in pathology?

A In pathology, yes

Q You view him as an expert in the pathology of lung diseases?

A Definitely.

Q Are you familiar with the name Kreyberg?

A Yes, he's a Norwegian, I think, he published some papers forty years ago, a Norwegian.

Q Do you view his works as authorities in lung cancer and its epidemiology?

A Kreyberg hasn't published in many, many years.

His major claim to fame, as I recall, is that he separated the squamous cell from the adenocarcinoma, but it was, I think, somewhat simplistic for the time.

There were other breakdowns that he could have made which were done later.

Q Are you familiar with Gio Gori?

A Yes, I am.

Q Do you view him as an expert in the epidemiology of lung cancer?

A Gio has published some papers on epidemiology. He hasn't done original work himself.

Q How about Peto and Doll, do you view them them as experts?

A Richard Peto and Richard Doll are two of the outstanding cancer epidemiologists in the world.

Q Are you familiar with the name Alvan Feinstein?

A Yes, I am.

Q He's a pathologist?

A He's a pathologist, yes.

Q He's an expert in pathology of lung disease?

A I don't know. Most of Feinstein's work is, they say, criticizing the work of others.

Q Do you question whether he's an expert in the field of lung pathology?

A I don't know if he's an expert in the field of lung pathology.

Q Are you familiar with Alfredo Morabia?

A Morabia, yes.

Q Who is he?

A As far as I know, he was a visiting scientist at Wynder, American Health Foundation and he worked there for a couple of years.

He did one paper which is based on cancer study data, which I made some tables for him, and he published a paper, sent it to me for review, and I told him not to publish it because it's based on too few cases.

Q How many cases were involved?

A The point he was making was that lung cancer in nonsmoking blacks was higher than lung cancer in nonsmoking whites.

It was based on less than ten cases of nonsmoking blacks.

So I told him, "Let's wait until we get more cases before we publish, with more follow-up," there would be more cases.

Q Do you view Alfredo Morabia as an expert in the epidemiology of lung disease?

A I wouldn't call him an expert, I would call him one of the people who works in it.

He is not very well-known and he's no longer with the American Health Foundation. I don't know where he is now.

Q Are you familiar with the name Michael Alavanja?

A Yes, he's at the National Cancer Institute, he's an epidemiologist.

Q Do you view him as an expert epidemiologist?

A Yes, I guess so. I don't know much of his work.

Q Did you ever hear of Joel Nitzkin?

A The first time I heard about it was in the letter that you sent.

Q That was sent in this lawsuit?

A Yes. I don't know him at all.

Q Have you ever seen him published anywhere?

A No.

Q Do you know most of the leading people on the subject of the risks of lung cancer?

A I would think I do, yes.

Q Have you ever heard of Samuel Hammar?

A No.

Q You have heard of Pelayo Correa?

A Oh, yes.

Q Who is he?

A He's an outstanding pathology and epidemiologist in New Orleans.

Q Have you heard of Darryl Carter?

A Carter, no.

Q Jay Lubin?

A Jay Lubin is with the National Cancer Institute. He's published a number of papers on the pathology of lung cancer cases in relation to other factors.

Q Do you view him as an expert in the field?

A He's a very good epidemiologist, yes.

Q Do you know Peter Lee?

A Yes, I do.

Q You have written with him?

A There was one paper that we did together.

Q Do you view him as an expert in the field of epidemiology?

A Peter has very little original data to work with, so it's -- he's more of a commentator on

other people's work.

He's a very smart guy, but I don't think he could be considered one of the leading epidemiologists, no.

Q You would not view him as an expert in the field?

A I consider an expert someone who has done original work and made his own interpretations of it, rather than interpreting other people's work.

Q When you say someone who has done original work, you mean someone who has conducted case control studies?

A Or cohort studies.

Q Is it fair to say the Surgeon General of the United States and his entire staff are not expert epidemiologists?

A Some of the people who -- many of the people who contribute to the Surgeon General's report are people who did the original studies and have sent their reports into the Surgeon General for compilation.

Q Let's break this down. There is no Surgeon General's report on smoking and health since 1964 that is itself based upon original research

done for the Surgeon General's office --

A It's all compiled data.

Q Let me just finish the question before you answer, because otherwise the record won't be clear.

The Surgeon General's reports are all based on compiled data, compiled by other people, is that correct?

A Right.

Q So it's fair to say that based upon the standards that you are using of what constitutes epidemiological expertise, the Surgeon General and his staff, in compiling the Surgeon General's reports, do not fit within the definition that you have given us?

A You are asking me what constitutes an outstanding epidemiologist.

I say that someone who simply comments on other people's work is not an outstanding epidemiologist. That differs from someone who compiles data and publishes, reviews articles, for example.

That's perfectly legitimate, but it's not responsive to the question you asked.

Q Let me see if I can then break this down.

Peter Lee, Dr. Peter Lee, has done some extensive work reviewing epidemiological studies conducted by others?

A Yes.

Q And he's a very bright man who brings considerable expertise in that field to bear?

A Right.

Q The Surgeon General of the United States and his predecessors have similarly compiled work of others?

A Let me try to elucidate the difference.

It is one thing to compile data relating to a certain field and publish it.

It's another thing to take the work of somebody and make your own interpretation, which may or may not be different from the interpretation of the original authors.

I think it's two divergent things.

Now, the question was asked in the context of who is an outstanding epidemiologist, and I still say you can't really be an outstanding

epidemiologist just reviewing the work of other people.

The Surgeon General's report is not a review, it's a compilation.

If Peter Lee simply compiled all of the data on a certain field and published it, that would put him in the same class as the Surgeon General's staff.

Q But what he did required greater analysis than simply compiling data, is that correct?

A It required analysis rather than compilation, yes.

Q So it was at a higher level of scientific inquiry than simple compilation?

A I don't know if I can say that.

Q You couldn't disagree with it, though?

A It's very hard for me to say -- to make an evaluation of that sort.

Q Just to close up this area, are you familiar with any Surgeon General of the United States since 1964 who you would classify as an outstanding epidemiologist?

A I say none of them are outstanding

epidemiologists.

As you very well know or should surmise, the Surgeon General's reports on smoking and health are compiled by the staff of his committee, and if you look at the front of every report there is at least sixty or one hundred people who have looked at all or some of the chapters and given their opinions on it.

MR. GROSSMAN: Why don't we take a break now. We can copy what you provided us and we will continue on from there, ten minutes.

(Whereupon, at this point in the proceedings there was a recess, after which the deposition continued as follows:)

Q Mr. Garfinkel, while we are waiting for Mark to finish with the copying, why don't we just get going.

Let's turn to the question of epidemiology generally.

You testified earlier that when you began with the American Cancer Society in 1947, epidemiology wasn't recognized as a separate field?

A I would think some people would call themselves epidemiologists, but it wasn't as well

recognized as it is today.

As a matter of fact, I have heard the statement made that epidemiology reached prominence in the minds of many scientists in the country through the American Cancer Society's epidemiological studies on smoking and health.

Q Epidemiology became a recognized academic discipline in the last couple of decades?

A Oh, yes.

Q Now, epidemiology has certain limits, is that correct?

A That's right.

Q One is that it's not personal, but rather is based upon population based studies?

A It's not clinical, it's based on population based studies, right.

Q It doesn't deal with the -- with efforts to determine the cause of disease in any individual, but rather with population groups, is that correct?

A That's correct.

Q In fact, you don't review individual cases of lung cancer to determine the cause of an individual's lung cancer, but rather you deal with

statistics regarding lung cancer in population groups in your field, is that correct?

A I wouldn't word it that way. I would say that we look at the -- at the relationship of certain factors in relation to the condition or the disease that occurs in an individual.

MR. GROSSMAN: Would you give that back to me, please.

(The answer requested was read back by the reporter.)

Q Now, it's a central tenet of epidemiology that population based studies do not prove causation in any individual, is that correct?

A You cannot prove causation from epidemiological studies alone.

Q You cannot prove causation in general from epidemiological studies alone and you cannot prove causation in any specific individual from epidemiological studies, is that correct?

A I would certainly agree with the latter, you can't prove it in any specific individual.

Let me amend what I said before.

I think you could, if there is a very

high relationship between cause and effect in an epidemiology study; for example, holding other factors constant, if you have all the subjects exposed to a certain agent getting the disease and none of the subjects who are not exposed to that agent not getting the disease, you get pretty close to causation.

Q You are talking now specifically of infectious agents primarily, is that correct?

A Well, this could happen with environmental agents, too.

Q If everyone who was exposed got a disease and no one who was unexposed got the disease, that by itself would tend to imply causation?

A It would be very close to causation. It would be nice to control other factors that may affect the result; for example, something like age, and it would be nice to have other evidence that links to this, and it also should be biologically a plausible cause and effect.

I am trying to marshal other evidence to help prove the cause and effect.

Q But insofar as epidemiology alone is

concerned, if the presence of an agent were shown to be both necessary and sufficient to cause the disease, then that would imply causation to you?

A Right.

Q Of course, smoking is neither necessary nor sufficient to cause lung cancer, is that correct?

A Lung cancer could occur in the absence of smoking.

Q Lung cancer occurs in the absence of smoking and it also does not occur in all smokers?

A Doesn't occur in all smokers.

Q So by itself epidemiology would not establish lung cancer, the lung cancer smoking link to you, is that correct, as causation -- let me rephrase the question.

A I am not going to give a yes or no answer to that, because in the case of lung cancer and smoking, what you have postulated may be true, but in the case of lung cancer and smoking, there have been so many studies which have all pointed to the same direction, with such a large relative risk, with a dose response, where the fact that people give up smoking, it reduces the risk.

That in itself, all those facts marshaled, even without the pathological evidence, would lead me to believe it's a cause and effect relationship.

Q Let me see if I understand --

A In other words, it's not, as you say, necessary sufficient evidence, it's marshaling all of the studies.

You get much more reliance in a cause and effect relationship, where the data is supported by numerous epidemiological studies, and where the relative risk is so high.

Q So high as it is for other cell and squamous cell carcinomas?

A All lung cancer, all lung cancer.

Despite the fact that one can say that in the great majority of cases epidemiology in itself cannot prove cause and effect, if I only had the epidemiological evidence that I cited, the high risk, the fact that there is a dose response, the fact that it goes down with the cessation of smoking, that it's true in many groups all over the world; all those facts in themselves, even without the other evidence, I would say proves cause and

effect.

Q Let's break this down into its component parts.

You are referring now to decisions about groups and not individuals?

A I am referring to groups and not individuals.

I am saying that the risk increases the more you smoke.

Q You are confirming that smoking is neither a necessary nor sufficient cause of lung cancer; that is, that lung cancer occurs in nonsmokers and it doesn't occur in all smokers?

A I am saying that that is true too, yes.

Q All right, we will talk later about some of the other things you just mentioned.

What is it, doctor, that elevates the aggregation of numbers in a survey into a science?

A Into a?

Q A science. What makes epidemiology a science?

A In order to be classified as a science, I think there are certain things that have

to be present.

For want of a better word, you have to apply the scientific method, which is a way of looking at data to eliminate any kind of obscurity of the basic result that comes out.

It's been said by some great philosophers that all of science is essentially measurement.

I think epidemiology embodies that concept.

Q So --

A Go ahead.

Q I'm sorry.

A I won't add to that right now.

Q The scientific method must be applied?

A Yes.

Q And the scientific method has been established in epidemiology?

A Good epidemiologists always use scientific methods to -- when they analyze their results.

Q When you say they use scientific methods, where could one find the scientific method of epidemiology?

A Where could one find it? I guess you could look in some epidemiology textbooks, which describe how to go about doing a study.

Q Is it fair to say that the scientific method in epidemiology begins with the generation of a hypothesis?

A Yes, you generate a hypothesis, then you test the -- as in all hypotheses -- the great probability is that the result is real.

Q As part of the scientific method of epidemiology there is a presumption of a null hypothesis?

A That's correct.

Q That means that unless it is demonstrated otherwise, there is a presumption that exposure to an agent was not the cause of a disease?

A You rule out that it was not the cause of disease and then accept the hypothesis -- the probability that it was the cause of the disease.

Q But there is a presumption?

A Let's not say cause, strictly speaking you are ruling out that it's not the cause and accepting the probability that it is associated with the agent in question.

Q There is a presumption of a null hypothesis and it is the --

A Negation of the null hypothesis that you are looking for in the final result.

Q In order to negate the null hypothesis, the burden is on the investigator to be able to meet certain criteria?

A Right.

Q One of those criteria is replicability, is that correct?

A No, replicability in my context is having other studies which show the same thing.

The criteria that the investigator looks at is that there are no other factors that may affect the result.

For example, if you are looking at cancer in cities versus countries, you wouldn't take sixty year olds who live in the cities and four year olds who live in the country.

You would try to equate age, because age is an important factor in disease.

You might also control such factors as where they work, marital status, which, of course, marital status is related to disease; you might

control a number of other factors, ethnic background and so forth.

Q Let's break this down into classifications of types of things that need to be excluded.

First, when I was referring to replicability -- I am glad that you corrected me -0- it is fair to say that that's a different criterion, is that correct, replicability?

A In my context it's duplicating one study with another; with different populations, let's say.

Q If the results of the study cannot be duplicated by other studies, the study cannot be confidently interpreted?

A It creates ~~sub~~doubt. It all depends on what we call the relative risk.

It is almost certain that if you take twenty studies and the real relative risk is let's say 1.3 or 1.5, there will be some studies which are adequately done which will not show an increased risk.

So if you don't have complete replicability, it does not necessarily mean that the

hypothesis is not true.

Q Replicability is the hallmark of scientific investigation, isn't it?

A Not in all cases, no.

Q Mr. Garfinkel, let's move on to the factors that you believe must be satisfied in order to negate the null hypothesis.

One factor that must be demonstrated is that the results of the study are not an artifact of chance, is that correct?

A That's correct.

Q And that is to say that it's up to the investigator to demonstrate that the results are statistically significant?

A That is correct.

Q What is statistical significance?

A Generally, the investigator will set up what is called a P value, a probability of let us say .05 or less.

That means that the probability of getting -- that the null hypothesis set up is that in 95 out of 100 chances -- 95 out of 100 trials, the null hypothesis will hold.

If five percent of all the trials --

hypothetical trials that are done, the result is -- the result shows that there is a difference in the agent versus the control, then one would say it is statistically significant, and reject the null hypothesis.

Q Now, in order to make epidemiology a science, there have to be certain conventions that are routinely applied, is that correct?

A Right.

Q And the convention that's routinely applied for determining statistical significance is a P value of .05 or less, is that correct?

A Or less, correct.

Q Sometimes a P value of .01 and sometimes even .001 is applied, is that correct?

A That's correct.

Q But at an absolute minimum, a P value of .05 is necessary for statistical significance?

A In some studies they have accepted a P value of .10.

It all depends on how certain you want to be of the result.

Q Well, in discussing the scientific method now and accepted criteria --

A The general convention is a value of P — of .05 or less, yes.

Q The general convention is a maximal P of .05 but often a lower P is necessary to draw a conclusion?

A It depends on the situation, yes.

Q And just to clarify the record on this, when a P is .05, that is the same thing as saying that there is a confidence level of ninety-five percent, is that correct?

A Right.

Q And for a confidence level of ninety-five percent to demonstrate statistical significance, both of the relative risks must be either in excess of 1 or below 1, is that correct?

A I'm sorry, would you reword that?

Q Yes. In order to demonstrate statistical significance, both the lower and upper limit of the confidence level must be under 1 or above 1?

That is --

A If it's above 1, then in saying that there is an effect; if the upper limit is below 1, then there is a less effect of the agent you are

looking for versus --

Q Just to clarify that, if both of the numbers are under 1, that means that there is a protective effect that is implied by the study?

A You don't necessarily call it a protective effect.

The factor you are looking at is statistically significantly less than the control factor.

Q Or to put it in other terms, the factor that is under investigation has a -- an inverse relationship to the disease being studied when both numbers are under 1?

A As compared to a controlled population, right.

Q Now -- so, we have established, Mr. Garfinkel, that under the scientific method of epidemiology, an investigator has the burden of demonstrating -- the burden of disproving the null hypothesis, and his first burden or one of his burdens in meeting that is to demonstrate statistical significance in his results?

A If he disproves a null hypothesis, he is establishing statistical significance.

Q He must establish statistical significance?

A That's the converse of what you are saying.

If he rejects the null hypothesis, he's saying that it is -- the finding is statistically significant, whether it's more than the control or less than the control.

Q I'm not sure I understand.

An investigator applying the scientific method -- what elevates surveys into a science -- cannot reject the null hypothesis if he does not come up with statistically significant results?

A All right, you could say that; I wouldn't word it that way.

There are two sides of the coin; you accept the null hypothesis, you don't accept the null hypothesis.

If you accept the null hypothesis, it's not statistically significant; if you reject the null hypothesis, it is statistically significant.

Q But you cannot begin by accepting or

rejecting the null hypothesis, it must be based upon some criteria, is that correct?

A I don't know what criteria you are talking about.

Q You said a moment ago that what elevates epidemiology into a science is having certain rules for the investigation?

A Right. Now I know what you mean.

Yes, you have to go according to certain criteria rejecting other -- controlling on other factors which may affect the result you are seeking.

Q One of the criteria that you apply to reject the null hypothesis is statistical significance so as to reject chance as a basis for the results, is that correct?

A No. I just explained to you if you reject the null hypothesis it is statistically significant.

It's not that you are looking for statistical significance. If you reject the null hypothesis, then the probability of such a finding is less than .05 and you are saying it's statistically significant.

Q I think we are saying the same thing, but I want to make --

A This is the disease rate in the control people.

The disease of the people you are investigating who have the exposure can come anywhere in between here and here, here, here, here, here, here (marking).

If it comes anywhere here, then you accept the null hypothesis case.

MR. COVERT: Wait a minute, let's identify it so we know what you are talking about.

MR. GROSSMAN: The court reporter won't be able to pick this up.

Maybe I can make it easier to be clear for the record.

MR. COVERT: He was drawing it. Would that clarify it better?

Q I think you were drawing --

A Let me try to say it this way.

If the finding in your study -- if in the finding in your study you accept the null hypothesis that there is no relationship, then it's not statistically significant.

If the finding is that you reject the null hypothesis because it's outside the five percent limits, then you are saying it's statistically significant.

It's like two sides of the coin, either you accept or you don't accept.

If you don't accept, it's statistically significant.

Q In order for -- I think it is two sides of the same coin, but just for the clarity of the record, in order for an investigator complying with a scientific method to reject the null hypothesis, the results of his study must be statistically significant?

A Yes.

Q Now, apart from statistical significance, to eliminate chance as a likely cause of results, an investigator must also demonstrate that bias in the study cannot explain his results, is that correct?

A Now, you can't really prove that bias doesn't affect results; you do the best you can to eliminate biasing factors.

But if there is some biasing factor

that you are unaware of that could affect the result, there is nothing you can do about it because you don't know what it is.

Q Just so this is clear, one of the factors that an investigator must inquire into in interpreting results of the study is bias?

A Right.

Q It's the burden of the investigator to attempt to demonstrate that his results are not explainable by bias, is that correct?

A Yes.

Q What kinds of bias may affect the results of an epidemiological survey?

A I mentioned several of them before.

Let me preface it this way. It's any factor which you are aware of that is related to the disease in question --

Q Let's --

A (Continuing) -- or the way you obtain information about the agent in question, that could also have some biases.

Q Just for clarity of the record; even if results of an epidemiological study are statistically significant, they may not show a true

association between exposure and disease because of the presence of bias, is that correct?

A The relationship that is statistically significant may be obscured -- the relationship may be obscured because of bias; it may either show that it is or is not significant.

Q So you may get false positives or false negatives because of bias?

A Right.

Q And the literature has defined many types of bias?

A Right.

Q One is called misclassification bias?

A Right.

Q What is misclassification bias?

A It's saying that someone, let's say, is a drinker when he's an ex-drinker or he's not a drinker when he is a drinker.

Q So the subjects involved may be misclassified and that's one source of bias?

A Right.

Q The controls may also be misclassified?

A Sure.

Q And that also would be a source of bias?

A Right.

Q The degree of exposure may be misclassified?

A The degree, the amount of exposure or the years of exposure may be misclassified; anything is possible in that sense.

Q And all of those could bias the results of an epidemiological study?

A Certainly.

Q Similarly, the disease that the person is diagnosed with could be misclassified?

A Could be misclassified.

Q That would also be a source of bias?

A That would be a source of bias.

Q We discussed intra-observer and inter-observer variability. That would be a source of bias?

A It all depends. If you have five people reading a certain slide, let us say, and four of them agree it's one thing and one disagrees with it, and the four are right and the other is wrong, hypothetically, and you accept the consensus view,

then there is no bias.

There could be an inter-observer variability without necessarily injecting a note of bias.

Q But it may be a note of bias?

A It may.

Q And, doctor, there have been studies of autopsies, autopsy results, that have demonstrated misdiagnosis of primary site by substantial numbers.

Are you familiar with those studies?

A Which ones are you referring to?

Q Are you familiar, doctor, with studies published by the Royal College of Physicians and Surgeons in London, finding forty percent misdiagnosis of primary site in lung cancer?

A No, I am not familiar with that at all. I would doubt very much if forty percent of lung cancers are misdiagnosed.

Q Are you familiar with studies published by Alvan Feinstein on misdiagnosis of lung cancers?

A Yes, I am familiar with some of his studies.

Q He has found in Connecticut hospitals misdiagnosis of primary site on the order of thirty to forty percent?

A I wasn't sure what percentage it was. I would doubt if it's thirty or forty percent.

We did a study based on -- Cancer Prevention Study I, in which we looked at the death certificate diagnosis of lung cancer and then compared it to what the pathologist said from the hospitals we got data from.

There was about a five percent underreporting and about a five percent overreporting, but the total number was about the same.

Q Let's break this down.

First of all, you are familiar with --

A If you want the reference to it, I will give it to you.

Q Let's just break this down into its component parts.

First of all, as far as Dr. Feinstein's studies are concerned, you are familiar with them, but you have not attempted to verify or confound the studies, is that fair to say?

A I am familiar with his paper, but I never tried to verify it one way or the other.

Q Now, his study was on misdiagnosis demonstrated by autopsy.

The CPS I study that you are referring to is a different kind of study, is that correct?

A It was a study of death certificates that we compared in the lung cancer cases to the pathologist's report.

Q That was a study simply to determine whether the doctor who wrote the death certificate properly indicated the diagnosis that had been made in the pathologist's report?

A Not necessarily, sometimes the pathology report comes after the death certificate is signed.

Q That study was not based upon autopsies, is that correct?

A Some of the pathology reports were autopsy -- some were surgical specimens, some were by autopsies.

Q Most of the death certificates in that study were not based upon autopsies, is that correct?

A I would guess the majority were not based on autopsies, right.

Q In fact, autopsies in the United States today are relatively rare, is that correct?

A This was thirty years ago, they weren't very rare then.

Q But certainly far fewer than half of the people who died had autopsies, is that correct?

A No, I will say it was more than half had autopsies in those days.

Q More than half of the individuals?

A Of course, it depends on the cause of death.

More than half of the cancers certainly would; probably less than half of the heart disease cases would.

Q Did the American Cancer Society maintain figures on the number of people involved in the CPS I study who had autopsies?

A I would have to get that paper.

We didn't look at all of them, -- where is my curriculum vitae? Everything is mixed up -- I don't have the paper here anyways. I will have to get this out.

I just don't recall what proportion. I know, as you say, the percent of autopsies back in the 60's was much higher than it is now, and it was higher in the cancers than it would be in the noncancers, because doctors were interested in finding the source of that, the source of the primary site.

In this particular study I am talking about, I can't really say what proportion was autopsies.

Q The CPS I study showed a five percent variance between the cause of death listed on death certificates and that found in pathology reports?

A Yes, but it was compensated by five percent lung cancers that were not on the death certificate.

In other words, five percent of those that said lung cancer were not lung cancer on pathology and five percent of those that didn't say lung cancer were lung cancer.

Q Now, did you check to see whether the overreporting of lung cancer was greater among smokers or nonsmokers?

A I don't think so; I don't remember.

Q Did you check to see whether the underreporting of lung cancer was greater among smokers or nonsmokers?

A I don't remember. I would have to look at the paper.

I don't think we did it, but we may have.

Q If the five percent variance were different between smokers and nonsmokers, that would constitute a bias in studies based upon death certificates?

A You are talking hypothetically. It might, it could work the other way, it could be more underreporting in smokers than under nonsmokers.

Q I may ask you a number of hypothetical questions during the course of this deposition, because that's the nature of the deposition.

A Yes.

Q But speaking hypothetically, if the numbers -- if death certificates of smokers overreported lung cancer and death certificates of nonsmokers underreported lung cancer, that would be a bias in lung cancer epidemiology, is that correct?

A That is true. By the same token, if

lung cancer in smokers was underreported and lung cancer in nonsmokers was overreported, that would also make a bias, it would make a larger relationship than it really is.

Q Are you familiar with studies that have been undertaken to determine whether death certificates tend to overreport lung cancer in smokers or nonsmokers?

A I don't know of any such reports; there may be, but I am not aware of them. I do have a table in one of my papers which tries to answer that question, but I can't remember the figures.

This is the paper we did on secondhand smoke, where we took 130 lung cancers as reported on hospital records, entered an independent smoking survey and an independent histological breakdown, and I could get that paper tomorrow, but I don't have it here.

Q Another kind of bias that's been noted in the literature has been referred to as recall bias?

A Right.

Q Are you familiar with that?

A Yes.

Q What is that?

A It is that a person can't remember what his exposures may have been thirty years ago.

Q For example, in environmental tobacco smoke, epidemiology recall bias may play a significant role, is that correct?

A It could in some people, and maybe not in the majority. I have no way of really doublechecking that. There is no way to doublecheck recall bias.

In other words, there is no written records of thirty years ago that would help you.

Q Similarly, in epidemiology regarding food consumption, fat consumption or vitamin consumption, recall bias may play a role?

A For some things it's much more difficult to recall to an interviewer what your exposure was; In others, it's much easier.

For example, in cigarette smoking, I would say it's much easier for people to recall if cigarette smoking happened than it is if food consumption happened thirty years ago, because people tend to smoke the same amount for many years.

Q It's easier -- you are suggesting it's

easier to measure cigarette consumption than certain other things that may be related to diseases?

A To certain other things, certainly, sure.

Q And among those other things would be exposure to ETS, fat consumption, vitamin consumption, vegetable consumption?

A It's much more difficult to recall a history of dietary consumption than it is cigarette smoke, I would say.

Q It's also much more difficult to recall history of exposure to environmental tobacco smoke?

A I'm not sure of that, I just don't know.

Q Haven't you yourself said that people don't consider the ventilation of the rooms they are in and similar factors when they try to determine the amount of -- let me finish the question -- try to determine the amount of environmental smoke that they may have been exposed to?

A It probably is more difficult to estimate environmental tobacco smoke; either now or in the past, certainly.

Q Now, in addition to what's been referred to as recall bias, the literature refers to something that's been called a wish bias?

Are you familiar with that? Ernst Wynder, among others, has written about that.

A Yes, Ernst has written about that.

I think what he was referring to was if some agent has been shown to cause disease, the wish bias would imply that somebody conveniently forgets that they were exposed to that agent.

Q People also -- I believe in Wynder's work, Wynder refers to the wish bias as indicating that some people, after being diagnosed with a disease, may recall greater exposure to environmental factors because they would like to blame others for those environmental factors?

A That might be true in some people, sure.

Q And all of these biases, regarding measurement of exposure, observer variability, misclassification, wish bias, could affect the results of an epidemiological investigation?

A If you put it in the subjective, could affect, one has to say yes.

Does it affect? In most cases, no.

Q It may or may not, and it's up to the investigator to apply scientific principles to determine whether bias is a likely cause of the results?

A Sure, in the case of smoking, for example, you could take all the unknowns, let us say, and assume they are all nonsmokers, instead of dividing them equally among smokers and nonsmokers, and still get a relationship.

In other words, that's the extreme. I have taken all the unknown biases and applied them, and you still get the effect, the same effect as taking the known.

Q And that's because of the size of the odds ratio?

A That's because of the size of it. That's why some people say that a relative risk, 0/- some people say two, some people say three, should be applied before accepting a result, because of some of these unknown biases.

Q That refers to the strength of association?

A The strength of the association,

right.

Q And before -- we really ought to turn to that later. But --

A Let me tell you about one other study that relates to this.

My colleague, Steve Stellman, did a paper a few years ago in which he took two subjects, the paper was called "Confounding," which is another way of talking about biases.

What other factors could have affected the result?

He took two items, oral contraceptives and breast cancer and bladder cancer and artificial sweeteners, and reviewed about five different studies.

In all these studies they published data in two ways; one directly without accounting for biases and next, in the same paper, taking into consideration aging and all the other things that we are talking about.

In every one of these studies, because there was a high relative risk, the same result came in whether you would take it into consideration, the confounding factors or not.

So we talk a lot about biases and confounding factors, but in at least two different areas of investigation it didn't seek to contribute very much to the interpretation of results.

Q That, of course, was where there was already a very high relative risk?

A Well, there wasn't as high as for cigarette smoking and lung cancer, but it was relatively high, two or three to one.

Q Do you recall what the relative risk was?

A No, I don't recall exactly.

Q Mr. Garfinkel, you just raised the term confounders.

Now, another form of bias that's sometimes separated out is confounding?

A Yes.

Q Could you state for the record what a confounder is?

A A confounder would be the same kinds of things I talked about before, age, ethnicity, place of residence; anything that has been suspected to be related to the disease in question.

Q Is it fair to say that a confounder is

a factor that may have a relationship with the disease in question, and that is more or less common among the disease group than the control group, but is unassociated with the --

A No, if they have a relationship to the disease in question, but you don't know if it's more related to the disease group or the control group; that's why you control for it.

Q You are saying that as part of the scientific method you try to control for all confounders?

A You try to control for the confounders, yes.

Q You try to control for all known confounders?

A All known confounders, that you can measure them.

There may be, as I said before, some confounders that you don't know about, or don't know how to measure.

Q Not every cause of lung cancer is known, is that correct?

A No, not every cause of other sites of cancer are known either.

Q When you said "no," the record may not be clear. It is correct?

A It is correct that not all causes of lung cancer are known.

Q Therefore, not all confounders for lung cancer are known?

A We probably could account for over ninety percent of all the causes of lung cancer.

If there are some other causes that may -- if there are some other agents that may cause cancer, lung cancer -- let me reword that.

If there are some other agents which may cause lung cancer which we still don't know, it could contribute very little to the relationship of lung cancer and disease; I mean agents and lung cancers.

Q At the individual level there are thousands of people every year in the United States who die of lung cancer, who have no known risk factors for lung cancer, is that correct?

A There are several -- probably several thousand that have no known risk, right.

Q That's every year?

A Right.

Q Since we don't know what the causes of their --

A Let me amplify that. Let me amplify that by saying this has to be a very small percentage of the total lung cancer deaths.

Q There are approximately 130,000 lung cancer deaths?

A 150,000, I think.

Q 150,000 in the United States, and 20,000 are among nonsmokers?

A Less than that.

Q Do you know the numbers?

A I think it's been estimated to be 15,000. Nobody knows for certain, because of all the people who die of lung cancer, we don't have their smoking histories, but based on epidemiological studies, we make some estimates.

Q Of those, what you are estimating is 15,000 lifetime nonsmokers who die of lung cancer every year many have no known risk factors for lung cancer?

A Some of them do, some of them don't, and those that have risk factors, among the risk factors that we know about are environmental tobacco

smoking, also known as passive smoking, radon exposure, asbestos exposure, radiation perhaps.

So a good portion of those who never smoked were exposed to one or more of these factors.

Q If there are unknown causes of lung cancer, that may account for the lung cancer of both smokers and nonsmokers -- let me go back.

There are unknown causes that account for the lung cancer of many nonsmokers?

A Of some nonsmokers.

Q Of some nonsmokers. There is no way of knowing whether those same factors may account for the lung cancer of some of the smokers?

A We have no idea even if there is such a factor that's causing it; that is, an external factor, may be genetic.

Q Or viral?

A Probably not.

Q But whatever the other factors are, whatever accounts for -- whatever unknown factors account for those deaths among nonsmokers may also account for some of the deaths among smokers, is that correct?

A It's possible.

Q You have listed among known potential confounders just a few. There are several others, aren't there?

A They don't come to mind right away. What are you thinking of.

Q Fat consumption?

A It's very doubtful that fat consumption could increase the risk of lung cancer.

I guess anything is possible, but I don't know of studies that have --

Q Alavanja?

A Alavanja, that was one study that was done; I don't know of many others.

Q Are you familiar with Ernst Wynder's cross-national studies --

A Cross-national, international studies?

Q Yes.

A Some of them, yes.

Q (Continuing) -- comparing lung cancer rates in the United States and other countries?

A Yes, lung cancer rates vary from country to country, certainly.

Q You yourself have published literature that has considered the lung cancer rates that vary

from country to country, is that correct?

A Among the publications of cancer facts and figures, there has been data that showed lung cancer rates in different countries.

Q In publishing your own articles on lung cancer and potential causes of lung cancer cross-nationally, you have relied on the World Health Organization statistics on lung cancer rates throughout the world?

A Which paper are you talking about? I'm not sure what you are talking about?

MR. GROSSMAN: Let's break for lunch. We will all be back in an hour, if Bruce isn't here in an hour, we will go on without him.

(Whereupon, at this point in the proceedings there was a recess, after which the deposition continued as follows:)

Q Mr. Garfinkel, we were talking about confounders a moment ago when we took a break.

In CPS II, which was a study that you were intimately related with --

A Yes.

Q A questionnaire was put together.

What was the purpose of that

questionnaire?

A Since we finished CPS I, there were a number of other factors that might be related to cancer that had come into prominence and we decided to investigate that.

Q The points that were on the questionnaire of CPS II were factors that were believed to have a possible relationship to incidence of cancer?

A Yes.

Q That includes lung cancer?

A Right.

Q How many questions were on the questionnaire?

A Oh, I can't remember exactly.

Q Approximately sixty-four?

A Sixty-four questions, no, it's many more than that.

CPS I we used to talk about 300 bits of information and the CPS II questionnaire had more.

It was somewhere between 300 and 400.

Q Among the things that were on CPS II were weight, is that correct?

A Yes. Height.

Q Sleeping patterns?

A Right.

Q Whether the person was married?

A Sure, marital status, ethnicity, there is the questionnaire.

History of cancer in the family.

Q How many --

A Medical conditions.

Q How many x-ray examinations they had had, whether they drank alcohol and how much?

A Right.

Q Socio --

A Eating habits, Page 3.

Q That included for fat consumption, fried foods, vitamins, beta carotene, leafy vegetables and you also wanted to know what occupations they had?

A Right.

Q Whether they were exposed to potential environmental hazards at work?

A Right.

Q All of these were believed to be potentially related to --

A Either related to cancer or other diseases or as a control factor.

For example, education, it's one of the possible confounders, so you put it in.

Q Education may -- education is one aspect of socioeconomic status?

A Right.

Q And socioeconomic status has been found to be strongly related to disease incidence, independent of other factors?

A I wouldn't say strongly related, but it's related to disease incidence.

Q Are you familiar with the name Marsha Angell?

A I think she's an editor of the New England Journal of Medicine.

Q The executive editor of the New England Journal of Medicine, well respected in the field?

A I don't know her personally, but I assume she's well respected in the field of editing.

Q The New England Journal of Medicine is certainly one of the premier medical journals in the United States?

A It has a very high reputation.

Q And among peer review journals it is at least as difficult as any to get published in?

A I am sure they reject many papers, some which may have merit because they don't have enough room for everything that is submitted to them.

MR. GROSSMAN: Let's mark this as Exhibit 4.

(The above described document was marked Garfinkel Exhibit 4 for identification, as of this date.)

MR. COVERT: I have an objection to that, it is not listed in the Pre-Trial Order.

MR. GROSSMAN: You never list cross-examination materials.

MR. COVERT: You don't.

(Discussion off the record.)

MR. COVERT: Objection.

MR. GROSSMAN: Mr. Covert has just objected to my use of Garfinkel Exhibit 4 on the ground that it isn't listed in the Pre-Trial Order.

I would note that neither was Mr. Garfinkel.

Q Mr. Garfinkel, let me hand you what has been marked as Garfinkel Exhibit number 4 for identification purposes.

Mr. Garfinkel, this is a copy of an article from the New England Journal of Medicine, July 8, 1993, it's an editorial by the executive editor on "Privilege and Health-What Is The Connection."

Have you ever seen this before?

A I don't remember seeing it; '93, no.

Q If I may direct your attention to the first page.

If you look at the third paragraph, it says after footnote 4, "The poor were nearly eight times as likely to be hospitalized for these illnesses," referring to asthma, diabetes "as were those with higher incomes and they had more severe disease at the time of hospitalization."

Then continuing it says, "So closely does socioeconomic status correlate with health that it confounds the interpretation of much clinical research."

Do you see that?

A Yes.

Q It says, "For example, studies of the effect of passive smoking on childhood asthma are uninterpretable until an attempt is made to control for socioeconomic status."

Continuing further on that page, it lists several other diseases that have been linked with socioeconomic status and it notes that socioeconomic status correlates more directly with health than do other well-known risk factors, including cigarette smoking."

Do you see that?

I will read it to you from the second paragraph on this page, it says, "The gap in mortality between the relatively advantaged and the disadvantaged is very large -- larger than the gap due to many other well-known risk factors, including cigarette smoking. And it has been growing wider."

Do you see that?

A Well, I can't accept that.

Q On what basis can you not accept that?

A It depends on what you are doing.

If you are comparing people who live in mansions and the higher socioeconomic status, who have very good medical background and compare it to

the people in the very lowest situation, who live in hovels, in skid row, who have all sorts of environmental conditions associated with living in that way, yes, you can get eight times as high.

Q Are you familiar with the report of Pappas from the New England Journal?

A I didn't read that report.

Q The same New England Journal of Medicine, July 8, 1993 contains a report by Pappas and others reporting that, "1986 Americans with a yearly income of less than $9,000 had a death rate three to seven times higher, depending on race and sex, than those with a yearly income of $25,000 or more."

MR. COVERT: Are you still reading from the editorial?

MR. GROSSMAN: Yes.

A I say to you that's not as big a variation of risk as heavy smokers -- not even heavy smokers, one pack a day or more smokers compared to never smoked.

Q Overall mortality?

A I am talking about lung cancer.

Q This is overall mortality?

A Overall mortality for people who smoke two packs or more a day is in that range; it's in that range.

Q You are saying --

A And a yearly income of less than $9,000 is very, very low.

Q A yearly income of $25,000 is not very, very high living in a mansion?

A That's not very, very high either, no.

Q Now, Mr. Garfinkel --

A Yes.

Q (Continuing) -- let's separate science from speculation.

A Right.

Q As a matter of science, there are many reports that show a significant disparity both in cancer mortality and in overall mortality between individuals who are low socioeconomic status and those who are high socioeconomic status?

A That's true.

Q And much of the difference cannot be explained by comparison of known confounders for the diseases in question?

A One of the confounders, of course, is

cigarette smoking, because people who are at low economic status have much higher smoking rates than high, recently it's been at least 2 to 1.

Q One of the confounders is certainly cigarette smoking and that's because cigarette smoking tracks with SES socioeconomic status, is that correct?

A Are you saying that the relationship is with cigarette smoking because cigarette smoking is a cause of death?

Q I am not talking about causes, I am talking about confounders.

If you were to --

A If it's a confounders, it's because it's an increased risk of death.

Q If you were to undertake a study of the effect of socioeconomic status on cancer incidence, one of the things that you would certainly control for would be smoking, is that correct?

A And why would you do that?

Q Because there are many studies that show a relationship between smoking and cancer incidence independent of other risk factors, is that

correct?

And for that reason you would certainly want to control?

A I would certainly want to control on smoking, if I was trying to find out the effect of socio status alone, I would probably control drinking habits, I would -- if I could, I would try to control on other factors having to do with air pollution, maybe take an index of air pollution in that area versus the area you are comparing it to.

Q Why would you do that?

A Because an index of air pollution might be related to disease.

I am not sure if it is, but it might be.

Q Are you familiar with incidence of cancer in Louisiana compared with the rest of the country?

A Louisiana has a higher incidence of lung cancer than many of the other states in the country, yes.

Q Are you familiar with cigarette consumption in Louisiana compared to other parts of the country?

A I am not sure.

I know that such data exists, but the CDC has gotten out some data on that, but I'm not sure.

Q If according to CDC numbers Louisiana ranked very high in lung cancer, but very low in cigarette consumption, would you conclude that there are other factors in Louisiana that are leading to the outbreak of --

A You can't make a comparison, an ecological study of that sort, there are a lot of different studies you can use to show that that comparison is not worthwhile.

Q What would you use?

A I would look at the death rate of cigarette smokers versus the death rate of nonsmokers in Louisiana.

Q If the incidence of emphysema -- is emphysema related to cigarette smoking?

A Very highly related.

Q Does it correlate to cigarette smoking at least as highly as lung cancer correlates to lung cancer in smokers?

A No, but it's highly related.

Q If the rate of emphysema in Louisiana were among the lowest in the country, would you assume based upon that number that cigarette smoking in Louisiana was at a low rate?

A No, because epidemiological studies, many, many epidemiological studies have shown that the relationship of emphysema to cigarette smoking is extremely high, 8, 9, 10 to 1.

Q So if there were a low incidence of emphysema that would suggest to you there was a low amount of cigarette consumption?

A Not necessarily.

Q What else would account for a low?

A Could be misclassification, could be a lot of things.

I would very much doubt that emphysema is related to cigarette smoking in other states and not in Louisiana.

Q What I am suggesting is, if in Louisiana the sale statistics show a low amount of cigarettes sold compared to other states --

A Yes.

Q (Continuing) -- and if the CDC numbers similarly show a low incidence of emphysema, would

you conclude on that basis that it's likely that there is a lower consumption of cigarettes in Louisiana than the rest of the country?

A I can't make that assumption.

Q How could you not?

On what scientific basis could you conclude that cigarette smoking in Louisiana --

A Well, because it's completely --

Q Let me finish the question.

If the U.S. Government sales data showed that Louisiana has less than average cigarette consumption per capita, and if the CDC numbers showed that emphysema in Louisiana is lower than in the rest of the United States, on what basis could you conclude, on what scientific basis could you conclude that cigarette smoking in Louisiana exceeds the national average?

A On what basis could I conclude that what?

Q That cigarette smoking in Louisiana exceeds the national average?

A I don't know if cigarette smoking exceeds the national average.

Q If, in fact, the government statistics

show that smoking in Louisiana is lower than the national average, you would have no basis to disagree with those statistics, would you?

A I don't know, I don't know how the data is collected. I would assume it's right, but I can't be sure.

Q If the CDC shows that emphysema in Louisiana is lower than the national average, you would have no basis to disagree with that, would you?

A Probably not.

I would have to look again at how the data is collected.

Q If the U.S. Government shows that cigarette consumption in Louisiana is lower than the national average and the CDC shows that emphysema is lower than the national average, those two data points taken together would tend to confirm that smoking in Louisiana is below the national average, is that correct?

A I don't know how the sample was taken, I can't say definitely that the conclusion you are going to has a scientific basis.

What I am saying is that you cannot

prove anything by an ecological comparison of that sort, you need direct epidemiological evidence.

If everything you say about Louisiana is true, it goes very much contrary to what the epidemiological evidence has demonstrated, so I would have to look more closely to see if that's real.

Q What I am asking, there is nothing about the ecological evidence that shows that if there is a low cigarette consumption rate there is -- that there should not be a low emphysema rate, is that correct?

A You are talking about ecological evidence, it could be almost anything, right, that's correct.

Q But if the sales numbers show low consumption of cigarettes in Louisiana and the CDC numbers show low incidence of emphysema in Louisiana, but also show very high incidence of lung cancer in Louisiana, it suggests that there may be other ecological factors working in Louisiana that account for the epidemic of lung cancer in Louisiana?

A The best I could say is that it may

be, that's all.

For example, I would want to look and see what the sales tax is in Louisiana, maybe people go across the borders to buy their cigarettes, I don't know.

Q But that would be speculation?

A That would be speculation, sure.

But what I am saying is by the scientific method which you want done you cannot make conclusions based on ecological evidence and the fact that all of the other evidence would indicate that cigarette consumption should be higher in Louisiana because they have a high lung cancer rate and because no other factors reasonably could account for that higher rate, reasonably could account for that higher rate.

Then I would say I would have to look at the data more carefully.

MR. GROSSMAN: Let's mark this as Exhibit 5.

A One of the other things -- may I add to that?

MR. GROSSMAN: There's no pending question, I'm sorry.

MR. COVERT: He can explain his answer any time he wants to, this is still applicable.

MR. GROSSMAN: I think he's just volunteering some new matter.

THE WITNESS: It's the same matter, if there's a lot of blacks, more blacks in Louisiana than some of the other states in the country, blacks do have a higher lung cancer rate and they smoke less than whites do.

Q So there are factors accounting for the lung cancer rates of blacks that cannot be explained smoking?

A No, it's explained by smoking, it's just that blacks smoke less -- fewer cigarettes -- more blacks smoke, but they smoke fewer cigarettes a day and that can account in part for the hypothesis that you just made.

Q If lung cancer rates in Louisiana are higher than the rest of the country among both blacks and whites, that would not account for it, would it?

A Depends to what degree it's higher in blacks or whites, may be a big differential there, I don't know.

Q Let me suggest to you that Louisiana has the fifth highest lung cancer rate in the United States of all the states, that it's in the bottom half of cigarette consumption and that it's forty-fourth in emphysema.

Don't those --

A What does that suggest to me?

Q Yes.

A I would have to look at the hypothesis that emphysema is underreported in Louisiana, that there may be a differential between black smoking and white smoking in Louisiana, and that it goes so contrary to the evidence from other sources that you can't take averages to make any conclusions.

Q What about the hypothesis that lung cancer is overreported in Louisiana?

A That is also possible, everything -- is it possible, sure.

I would think because of the long history of the Ochsner Clinic attracting people -- you see that's another possibility that we haven't talked about.

Somebody lives in Texas and he hears about the Ochsner Clinic and other hospitals that

are well versed in taking care of lung cancer patients.

He gets to a hospital in Louisiana, he's not included in the sample because he doesn't -- of smokers, because he doesn't live in Louisiana, but yet his death is counted as a Louisiana resident.

Q How do you know about that?

A I don't know, I am hypothesizing, just like you are.

Q Purely speculation?

A Just like you are speculating.

Q But I am asking you whether the large relationship -- let me start that again.

I am asking you whether the large disparity between lung cancer rates and emphysema rates in Louisiana may be accounted for by environmental factors present in Louisiana?

A In my opinion there is not enough evidence from other studies on any environmental agent that is so widespread that it could cause an increase in lung cancer that can account for this kind of difference.

Q Have you read the studies of Maurice

Gottlieb --

A I have read some of the studies.

Q (Continuing) -- on southern Louisiana?

A Gottlieb never controls on smoking.

Q Have you read -- never controlled on smoking?

A Doesn't control on smoking, because he can't.

Q Are you familiar with an EPA official named Jeff Bouvier?

A No.

Are you familiar with a paper produced by Kyla Hammond looking at lung cancer rates in areas that have high indexes of air pollution versus those that don't, and controlling on smoking on age which nobody else has been able to?

Q I am. But in this process -- I know you haven't been in a deposition before -- the only one who asks questions is me.

A Well, that's sort of an answer it's a rhetorical question.

MR. GROSSMAN: Let me have marked, if I may, what we will mark as Exhibit number 5.

(The above described document was

marked Garfinkel Exhibit 5 for identification, as of this date.)

Q Mr. Garfinkel, let me hand you what's been marked for identification purposes as Garfinkel Exhibit number 5.

I will just note for the record that it is a section from a book called, "Fundamentals of Surgical Oncology," edited by Robert McKenna, containing a chapter called "Cancer Epidemiology" with your name on it.

A Right.

Q If you look at Page 29, you say that, "Comparison of cancer deaths among various countries may provide valuable clues about possible environmental factors responsible for certain diseases"?

A Right.

Q "For example, the mortality rate for stomach cancer is about eight times as high in Japan as in the United States, and that of breast cancer is five times higher in the United States than in Japan."

A Yes.

Q What does that suggest to you?

A It suggests that the area where one might start an epidemiological study for certain hypothesis that might explain this difference.

Q Now, the differences are clearly not purely ethnic or racial, is that correct, because second and third generation Japanese in the United States adopt a health profile that's very similar to other Americans?

A The breast cancer rate in second generation Japanese approaches that of Caucasians more than Americans, but it is not as high.

Q What about the lung cancer rate of Japanese Americans, second and third generation?

A I'm not sure, I don't know of any study that -- maybe there are studies in Hawaii, but I'm not familiar with them.

Q On the next page, on the following pages, on 30 and 31 you have, "Age-adjusted Death Rates per 100,000 Population for Selected Cancer Sites for 48 Countries."

A Yes.

Q That's based upon World Health Statistics Annual?

A It is indexed on the World Health

Statistics Annual 1980 to '82.

Q World Health Statistics Annual is published by who, the World Health Organization?

A World Health Organization.

Q Those are the figures generally relied upon by epidemiologists and others in your field to compare cross-nationally cancer incidence rates?

A Cancer mortality rates.

Q Cancer mortality or incidence rates?

A The incidence rates comes from a different book called Cancer Incidence in Five Continents or something of that sort.

It comes from a different volume.

Q Have you ever attempted to compare the lung cancer mortality rates on your table here with smoking incidence rates in the countries involved?

A Again, that's the kind of an ecological exercise which most epidemiologists don't think -- could only give you clues, but can't prove cause and effect.

Q It can help to generate hypotheses?

A It can generate hypotheses.

Q You are familiar with the work of Ernst Wynder in this area where he compares the

smoking rates in various countries and the lung cancer mortality rates in those countries?

A I am not sure which paper you are referring to.

Q Are you familiar with a paper of Wynder, Taoli and Fujita from the Japanese Journal of Cancer Research entitled "Ecologic Study of Lung Cancer Risk Factors in the United States and Japan"?

A No, I am not familiar with that paper.

Q Are you familiar with a magazine called "Cancer"?

A I am an advisory editor of the journal called "Cancer."

Q Are you familiar with an article by Wynder, Fujita and several others, including Kuryama called "Comparative Epidemiology of Cancer between the United States and Japan"?

A No, I am not familiar with that.

MR. COVERT: What is the date of that?

MR. GROSSMAN: That is 1991.

Q Doctor, is it fair to say that you have not attempted to make any study of the consumption of cigarettes versus the incidence of lung cancer or mortality from lung cancer on a

cross-national basis?

A I have never done any study of that sort.

Q If studies of that sort showed that men in Japan have for the last five decades smoked at rates approaching twice those of the United States, smoking on average cigarettes of higher tar and nicotine content and smoking the same or a greater number of cigarettes per day, but that the mortality rates from lung cancer in Japan, from lung cancer, were one-third those of the United States, would that generate a hypothesis to you that there were other ecological factors that accounted for the difference in lung cancer between the two countries, apart from smoking?

A I, at present, just listening to those figures, do not have any hypothesis that I would test, maybe I would test dietary habits, it's the only one that comes to mind.

Q Would you test fat consumption?

A As part of dietary habits, sure.

Q Are you familiar with a paper by Ernst Wynder that found that lung cancer incidence on a country by country basis was directly proportional

to fat calories loss statistics?

MR. COVERT: I am going to object to this line of questioning.

You are saying what Wynder said, if you have the document, I think it would be only fair to let Mr. Garfinkel look at it.

Q If Mr. Garfinkel is familiar with it, I will --

MR. GROSSMAN: Well, why don't we mark it, this is Exhibit number 6.

A If I read that article I don't recall it, but what I would say is it's true of this and other ecological studies.

All it could do is generate hypotheses. Hypotheses that have to be tested.

There is a paper by Carroll, I think his name was, which showed dietary fat and colon cancer, dietary fat and breast cancer, and there was an almost perfect correlation between one and the other.

That itself does not prove that dietary fat has anything to do with these two cancers, it just means that you could then start a study to try to show whether or not this is true.

One such study was started in Massachusetts and they found within a group of nurses, 80,000 nurses, something of that sort, that there was no relationship between percent of diet consumed of fat and breast cancer.

Q Now we are talking about lung cancer, let's mark this as Exhibit 6.

A But what I am saying is even though the ecological study showed that there was a relationship between countries, when you did an epidemiological study it turned out in this particular case that the hypothesis was not proven; the same thing can be true of lung cancer.

Q You are familiar in dietary fat studies and lung cancer with the Alavanja study?

A Yes.

Q Are you familiar with any others?

A Yes, we talk about diet now, the American Cancer Society has done some studies of diet and colon cancer; are you speaking only of lung cancer now?

Q On lung cancer.

A Dr. Hirayama has done studies of lung cancer.

Q You would call --

A Would not.

Q You would call Ernst Wynder --

A Ernst Wynder has a very formidable reputation --

Q He is one of the outstanding --

A (Continuing) -- and widely published.

Q He is one of the outstanding epidemiologists in the country?

A I think most people would classify him as an outstanding epidemiologist.

Q Now, turn to Exhibit number 6, which was published in the Journal of the National Cancer Institute, that's a well respected journal?

A Sure, yes, 1987.

Q In 1987.

If I may direct your attention first to the abstract, do you see it says that, "A regression analysis weighting each country by the square route of the appropriate population and adjusting for several covariates showed that calories from dietary fat were highly significantly associated P.0001 with lung cancer mortality. This finding was obtained after accounting for

disappearance data for tobacco P.0001. The dominant factor for lung cancer and total nonfat calories P.002."

Q Do you see that?

A Yes, I see that.

Q Let me direct your attention to Page 635.

MR. COVERT: Mr. Garfinkel was kind of perusing, would you give him a chance to look at it.

MR. GROSSMAN: If you want.

A Okay.

Q The right hand column, three paragraphs down, "An interesting observation" --

A (Continuing) -- "of this study is that fat calories" --

Q Could you read that into the record?

A "An interesting observation of this study is that fat calories are more strongly associated with male lung cancer mortality than are tobacco-disappearance data. The skewness of tobacco-related exposures greatly handicaps our efforts to describe a population-base summary statisticd to use in analysis. We therefore relied on mean values to describe relevant exposure."

Q Now, Mr. Garfinkel, I take it that it is your testimony that such a finding would not prove a cause and effect relationship between ingestion of dietary fat and lung cancer, is that correct?

A This is an ecological study and therefore my same principle applies.

Q But it would generate a hypothesis that fat consumption may be a cause of lung cancer, is that correct?

A A hypothesis that fat consumption may be a cause of lung cancer, yes.

Q And that could be tested by case control or cohort studies?

A Yes.

Q Now the only case control or cohort study that you are familiar with on ingestion of dietary fat and lung cancer is the Alavanja study?

A That's the only one I know of, yes.

Q In the Alavanja study of several hundred Missouri women who had never smoked, consumption of dietary fat was significantly associated with lung cancer, particularly adenocarcinoma, is that correct?

Q And fat consumption?

I am talking about fat consumption and lung cancer.

A No others come to mind, I am sure there have been others.

Q Let's just go over this one confounder for a moment --

MR. GROSSMAN: Could we mark this as Exhibit number 6.

(The above described document was marked Garfinkel Exhibit 6 for identification, as of this date.)

Q Mr. Garfinkel, let me hand you what has been marked for identification purposes as Garfinkel Exhibit 6, that is an article by Ernst Wynder, James Hebert and Geoffrey Kabat.

Now are you familiar with Dr. Hebert?

A Yes, I met him.

I know Geoffrey Kabat very well.

Q Geoffrey Kabat is an expert in epidemiology?

A He's an epidemiologist who has published some papers. I wouldn't call him one of the outstanding experts in the country.

Q You would call --

A Would not.

Q You would call Ernst Wynder --

A Ernst Wynder has a very formidable reputation --

Q He is one of the outstanding --

A (Continuing) -- and widely published.

Q He is one of the outstanding epidemiologists in the country?

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A (Continuing) -- "of this study is that fat calories" --

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A Yes.

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A That's the only one I know of, yes.

Q In the Alavanja study of several hundred Missouri women who had never smoked, consumption of dietary fat was significantly associated with lung cancer, particularly adenocarcinoma, is that correct?

A I would have to refresh my memory about that study, I don't recall it.

MR. GROSSMAN: Let's mark this as Exhibit number 7.

(The above described document was marked Garfinkel Exhibit 7 for identification, as of this date.)

Q Mr. Garfinkel, let me hand you what has been marked for identification purposes as Garfinkel Exhibit 7, which is a copy of the Alavanja study, Alavanja and others, including Ross Brownson, from the Journal of the National Cancer Institute in 1993 entitled, "Saturated Fat Intake and Lung Cancer Risk Among Nonsmoking Women in Missouri."

I would like to direct your attention, if I may, to the discussion, which is on Page 1911?

A Before you do that, let me just read the background again.

What page do you want me to look at?

Q Page 1911.

Let me apologize for the xerox that we have here, I wish that everything came out a little cleaner and clearer in the picture, but if you can turn your attention to the discussion, Page 1911, in

the right-hand column the last paragraph it says:

"In our study of nonsmoking women, the effect of saturated fat intake was most pronounced for lung adenocarcinoma with an 11-fold elevation in risk in the highest versus lowest quintiles of saturated fat consumption."

Do you see that?

A Yes, just a moment. Well, looking at the confidence limits for that in Table 7, I would say that is based on very, very few cases and you can't really take the 11 to 1 effect very seriously.

Q Let's see what the confidence limits are?

A It's 3.77 to 34.4. So if you look at the lower limit --

Q The lower limit is 3.77?

A Yes.

Q Even if one were to accept the lowest limit of confidence that's a strong --

A It may look like it, but it's based on a very small number.

Q Let me finish my question before you answer.

Even if one were to look at the lowest

limit of the confidence level which is 3.77, that would indicate a strong association between dietary fat consumption and lung cancer, is that correct?

A I would want to know how many cases it's based on.

Despite the fact that it is statistically significant, according to the general thing, if this is based on six cases or eight cases, I can't take this very seriously.

Q Could you look at the first page, it's based on 429 case subjects?

A Adenocarcinoma is based on 211 cases but we split that into five different groups and looking at the confidence limits, we know that the smallest group is the ~~highest~~ number five.

I can't say how many there is, but if it splits equally, five, there may be forty on the average and I would say that the highest is probably ten or less than ten.

Q And the second highest shows a relative risk of 4.3 which was also statistically significant?

A That's based on a larger number.

Q Also statistically significant?

A Yes, with a lower limit 1.6 to 11 -- it's still not a very large number.

Q And the middle quintile shows a -- an inflated relative risk which is also --

A The biggest number according to the confidence limits, I wish people would write numbers in here, is quintile number 2, that's a very small span for the confidence limits, much smaller than any of the others.

Q Let me see if I understand this.

You are saying that the largest number of cases fall in quintile number 2, is that correct?

A It may even be in quintile number 1, but I would think it would be quintile number 2.

Q Now, as I read this chart, and tell me if I'm wrong, as compared to the lowest level of fat consumption --

A Right.

Q (Continuing) -- lowest quintile of fat consumption, both the second quintile, the third, the fourth and the fifth all show elevated rates of lung cancer, is that correct?

A Yes, and it also shows a dose response, which is very important.

Q First of all, all four of those quintiles show an elevated risk, correct?

A Right.

Q Secondly all four of those quintiles are independently statistically significant?

A Yes.

Q Third, there is a clear dose response, is that correct?

A Right.

Q All of those would indicate to you a causal relationship between fat consumption and adenocarcinoma?

A It would appear that there is a causal -- a very strong association.

Q There is a very strong association?

A Right.

Q Now why would you say there is a very strong association but hold back on the question of whether there is a causal relationship?

A Two reasons, I want to see the numbers involved here. For all I know out of the 211 cases, there may be, it's not likely, but it could be ninety in the lowest category and sixty in the next category and then even though the others are

statistically significant, they may be based on very small numbers.

So I would want to see that.

Q At a minimum, given the fact that the Wynder-Kabat study demonstrated on a cross-national basis that lung cancer mortality rates were associated with fat consumption and that that tends to be confirmed by a case control study, showing this level of relative risk, one would have to control carefully for saturated fat consumption in --

A No.

Q Let me finish the question.

A I'm sorry, go ahead.

Q Let me start it again.

Given the fact that Wynder and Kabat study has shown a relationship between fat consumption and lung cancer mortality rates on a cross-national basis and that Alavanja's case control study has demonstrated an association between dietary fat consumption and adenocarcinoma at the levels reported, it would at a minimum indicate to you that there is a substantial likelihood that fat consumption may be related, and

may be a causative factor in lung cancer?

A May I answer that?

Q Yes.

A I would not like to draw any conclusions based on one case control study.

If I could find the same thing in the women's health study, the nurses' health study, I am sure they must have looked at this, but I can't be positive.

If I find it in two or three studies, I would have much more confidence that such a relationship exists and is real.

Q Could you look at Page 1911?

A Excuse me, I am not finished with my answer yet.

As an example, about ten years ago Jeffrey Howe in Canada did a study on saccharin consumption and bladder cancer.

He found it was related in men and not in women. There was also some animal evidence that indicated that saccharin consumption increased the risk of cancer in experimental animals.

This created quite a stir.

Since then a number of other people

have tried to duplicate his finding. In all, as I recall, there were ten other studies that were done, case control studies that were done on ~~fat~~ consumption -- sorry, that's saccharin consumption and bladder cancer.

None of them found a relationship and at a meeting I attended Jeffrey Howe said, "Look, I found it, I did it as best I could, it was a well-designed study, but mine showed it, the others didn't. I have to accept the fact that there is no relationship."

Another example --

A Brian McMahon found a relationship between coffee drinking and pancreas cancer.

Subsequently a number of studies found no such relationship.

Therefore, most epidemiologists do not accept the fact that there is a relationship between coffee drinking and pancreas cancer.

So I adopt the same thing to this.

If I can see three or four other studies which show me that there is a relationship I will accept it, if some of them show no relationship, I would have some doubts about

accepting the significance.

Q Mr. Garfinkel, could you look at the first two sentences after the word "Discussion" on Page 1911 of the Wynder article that has been marked as Exhibit 6?

A "Five case-control studies and one cohort study have reported a positive association between the risk of lung cancer and a diet high in fat or cholesterol. In the two earlier earlier case-control studies, both from Hawaii, an effect of cholesterol intake was seen primarily in male smokers were squamous cell carcinoma."

Q The next sentence.

A "Recently, however, a reanalysis of one of these studies found that both men and women smokers who ate foods high in fat content were at a significantly elevated risk of lung cancer an odds ratio of 3.3 for highest versus lowest quartile of high-fat dessert consumption among women."

Q So there have been -- apart from Alavanja, which had not yet been published, there had been five case control studies and one cohort study and all showed a relationship between fat consumption and lung cancer, so I guess it's been

proved that fat consumption causes lung cancer?

A I want to look at these studies again I am not familiar with these studies, I haven't followed them, so I will want to look at them.

Q Let me just see if I understand you, then we can move away from this area.

First, you said that you will not by itself trust what you referred to as an ecological study.

What is an ecological study?

A An ecological study is when one tries to make a correlation between an index of some kind of exposure at incidence or mortality rates for some type of cancer.

Q What is the index and exposure that you are referring to?

A It might be an average amount of fat consumption in a population, in a country.

MR. SHEV: Could you read back the last two questions and answers.

(The portion of the record requested was read back by the reporter.)

Q Mr. Garfinkel, you testified that an ecological study could generate a hypothesis, but

not demonstrate a cause and effect relationship.

And you would look to case control or cohort studies to that?

A Right.

Q I then pointed you to Alavanja, the Alavanja study, and you said that alone could not demonstrate a cause and effect relationship, notwithstanding its strong dose response finding and statistically significant result because you would want to see it replicated by other studies?

A Right.

Q Two or three other studies.

Then when it was noted that there have been five other case control and one cohort study, you said it still would not be proved?

A No, I didn't say that, I say I would want to read those studies and see how well they were done, what they were measuring, et cetera.

Q So it is not proved to your satisfaction?

A If I read all the studies and it seems to be well done, with enough cases, I would say that there probably is -- very likely that there may be a cause and effect relationship between the

epidemiology between fat consumption and lung cancer. It may be in nonsmokers alone, I don't know or maybe smokers, I would have to read the other studies.

Q Some of the studies were among smokers and some were among nonsmokers?

A I would have to look at it.

MR. GROSSMAN: Let's take a short break.

(Discussion off the record.)

MR. GROSSMAN: Mark this as the next exhibit, 8.

Before you do it then let me just note for the record that when we were off the record I suggested that although I have no problem with Mr. Garfinkel giving many examples after a simple yes or no response and raising things about saccharin and other kinds of studies, but this is a time consuming procedure that way.

Okay, Mr. Garfinkel, there was something else you wanted to add to the record?

THE WITNESS: Yes, I wanted to add one other comment on the Wynder paper, "Association of Dietary Fat and Lung Cancer," in the first sentence

of the discussion, for the record, we should note that he says, "In interpreting these results" the results of ecological studies -- "it must be emphasized that cigarette smoking is the principal cause of lung cancer. In the absence of tobacco smoke, lung cancer will generally be rare, regardless of the diet consumed."

In the Alavanja paper, it occurred to me in looking at this and and I have to read this more carefully, that the correlation with saturated fat might be because there is a negative correlation between vegetable and food consumption and saturated fat consumption.

Now, we don't know which is the primary factor but we know both these items are inverse related.

The more saturated fat you eat the less fruits and vegetables.

So I would have to look at this carefully to see if they controlled, that might be a possible confounder.

Q So you are saying, Mr. Garfinkel, that diet would clearly seem to have a relationship with lung cancer incidence among both smokers and

nonsmokers and whether it's fat consumption or fruit and vegetable consumption, that's the principal component of that, is something that's up in the air to you?

A I would have to look at the studies further and see if that was controlled for, yes.

Q But diet in one respect or another clearly has a role in lung cancer etiology both in smokers and nonsmokers?

A It may have an effect, but I would quote Dr. Wynder as saying whatever effect it is, it must be really small compared to the effect of cigarette smoking.

Q Let me hand you what has been marked for identification purposes as Garfinkel Exhibit 8.

Could you confirm that that is a copy of the questionnaire for men from the CPS II study of the American Cancer Society?

(The above described document was marked Garfinkel Exhibit 8 for identification, as of this date.)

A That seems to be -- yes, it is.

Q Now you testified earlier that the questions contained on this questionnaire were

largely to -- were largely based upon what had been identified as potential risk factors for lung cancer in the population, is that correct?

A Oh, no, there is potential risk factors and control factors for cancer and for other diseases, too, not just for lung cancer.

Q Including lung cancer?

A Including lung cancer.

Q For example, weight has been related to cancer and lung cancer in particular, is that correct?

A Weight has been related to cancer, yes.

Q People who are significantly overweight or significantly underweight have a higher incidence of lung cancer after controlling for other factors, is that correct?

A We found that to be true, that the relative underweights and overweights as higher for total mortality, it wasn't higher for lung cancer.

Lung cancer rates were lower in underweight people.

Q Significantly underweight?

A For those people who weighed less than

eighty percent of the average weight for people of their same age, height and section, the lung cancer -- the lung cancer rate was lowest of all in the categories.

Q How about those people who were forty percent over average?

A Lung cancer rates were higher but not significantly higher.

Q People who have abnormally long or abnormally short sleeping patterns have a higher incidence of cancer, is that correct?

A As I recall, the lung -- yes the long and short sleeping patterns, sleeping patterns are associated with a higher lung cancer rate.

Q People who are married are less likely to get cancer than people who have never been married, is that correct?

A Slightly less likely, yes.

Q People who have high alcohol consumption are more likely to develop cancer than people who have fewer than two drinks per day, is that correct?

A Depends on what cancer you are talking about.

For those who -- for cancers of the upper respiratory tract, esophagus, throat, larynx, the rates are considerably higher, maybe six times higher for the heavy drinkers for breast cancer it was about 1.2 -- the relative risk was about 1.2.

Heart disease it was actually lower for the heavy drinkers than nondrinkers.

Q Are you familiar with Dr. Correa's study on the effect of alcohol and the risk of lung cancer, Stephanie Correa and others?

A I am not sure if I remember that paper.

Q Are you familiar with papers that have found a positive correlation between excessive alcohol consumption, particularly beer consumption and lung cancer risks?

A I believe I remember one paper on that, yes.

Q You are not familiar with any study that's shown that people who drink four, five, six drinks a day have a lower incidence of lung cancer than people who don't drink, have you?

A I haven't seen any paper of that sort, it may be.

Q We have already gone over that high fat foods, fried foods, lack of vitamins, lack of leafy vegetables have all been associated in some studies with higher lung cancer risks?

A Lack of, yes.

Q And certain occupations are related to higher lung cancer risks?

A Asbestos workers, roofers, people who stand by asbestos operations, yes.

Q People with lower education and socioeconomic status are associated with higher lung cancer risks, is that correct?

A In all of these cases, one has to look at other factors which may be associated with it.

Q Well taken.

Now, have you done any studies to determine the correlation between people between the question whether people smoke and the question whether people have these other potential risk factors for lung cancer. Have you run any such studies internally?

A Would you give me an example of what you are talking about?

Q Yes, are smokers more or less likely

to have heavy alcohol consumption than nonsmokers?

A Most studies have shown that people who drink are generally heavier smokers than people who don't drink, and there is a correlation between numbers of drinks and numbers of cigarettes smoked.

Q Have you run figures based on the CPS II study to determine the correlation among the group study?

A Not in relation to lung cancer.

Q In relation to total mortality?

A We did it for breast cancer, we looked at the total breast cancer by drinking and also for the nonsmokers.

So in that sense we looked at it.

Q Have you looked to correlate to see whether -- let me start that again.

Have you looked to see whether smoking correlates with these other risky lifestyle patterns based on your CPS II survey?

A I can't answer you offhand without looking -- thinking about it.

I just don't recall.

Q Have you seen published studies that suggest that smokers are more likely to come from

lower socioeconomic groups, more likely to engage in occupations where they will have exposure to carcinogens, less likely to eat leafy vegetables, less likely to take vitamins, more likely to eat fried foods, more likely to eat high fat foods, more likely to drink alcohol in large amounts, less likely to be married, more likely to have unusual sleeping patterns and more likely to have weights that are either --

A We haven't looked at all those factors.

I know we have looked at smoking in relation to dietary consumption of leafy vegetables. I forgot what else we looked at in that particular paper.

Q What did you find?

A Smoking is negatively correlated with consumption of leafy vegetables.

It's positively associated with fat consumption.

People who live in -- people who have lower socioeconomic status tend to smoke more than people who have a higher socioeconomic status, they tend to -- they are less likely to quit smoking.

So that's some of the things that you are talking about.

There may have been others, I don't recall.

Q All right, so what you found was with regard to those risk factors that you have just identified, people who smoke are more likely to have the other risk factors for lung cancer as well, is that correct?

A Put it the other way; I would say that one of the reasons why people who have these attributes have higher rates of lung cancer is that they smoke.

Q But that's not the question.

A We didn't look at these things independent of smoking.

MR. GROSSMAN: Would you please repeat the question.

(The question requested was read back by the reporter.)

A Yes, I answered yes.

Q Now, you just said a couple of moments ago that you didn't look at these additional factors independent of smoking.

That is, you didn't look in your CPS II study to determine whether fat consumption was a confounder for the relative risk numbers that you generated for smoking and lung cancer?

A I'm sorry, repeat that?

Q When you generated relative risk numbers for smoking and lung cancer --

A Right.

Q (Continuing) -- did you control for fat consumption?

A In the latest study we didn't. In an earlier study we did control on many, many factors.

I think the best way to answer your question and to give you some advice on how to look at this is that in CPS I, we looked at people who never smoked and people who smoked one pack a day of cigarettes.

This is a study based upon, as you know, one million people.

In that case, because of the size of the study, we were able to compare nineteen different factors of the nonsmoker or the matched smoker, this included sleeping habits and age and dietary habits and height and even things that

didn't make any sense.

Nineteen different factors, including most of those that you listed here.

We found that the cigarette smokers -- so we controlled as best you can, not statistically, but directly, and we found that the cigarette smokers had twice the death rate of the nonsmokers, and they had over ten times the rate of lung cancer.

I don't know how much more you can take these other factors in consideration.

Q In CPS II did you control for these other factors?

A We didn't have to. We proved from CPS I that we -- if you do a matched study you can find the same relationship holds than if you don't match on these other factors.

Q The same relationships, that is, you end up with the same relative risk?

A As a matter of fact, in CPS II the relative risk is higher than in CPS I.

Q I know it's higher than CPS I. What I am asking you is if you control for these factors, do you end up with the same relative risk as if you don't control for these factors?

A If you don't control for the factors, instead of being 22 to 1 for males, it might be 19 for 1 or 23 to 1.

Q But you don't know what it would be?

A It's a tremendous relationship.

Q Please answer the question. Do you know what it would be if you control for these factors?

A We didn't do it, so I can't tell what you it would be.

All I can say is that it would be very, very little difference from the overall risks, uncontrolled.

Q Let's just --

A And I base this on what we found in the first study.

Q There are two things that can be said. One is you didn't control for the factors, correct?

A We always control for age, and we controlled in some cases for years [illegible] number of cigarettes smoke per day, [illegible] so forth.

Q You controlled for number of cigarettes smoke per day, but for the non-cigarette factors such as sleeping patterns, alcohol

consumption, fat consumption, fried food consumption, vitamin consumption, leafy vegetable consumption, occupational exposure and socio-economic status --

A In our CPS II study --

Q Let me finish the question.

A Risk factors in women, we did not control on those things.

Q And all of those factors, in all of those factors, the unhealthy profile corrolates with smoking?

A Yes.

Q So if the unhealthy factor corrolates with smoking and you did not control for those factors, the failure to control would elevate the relative risk, wouldn't it?

A I am going to say no, and the reason I say no is that in our first study the relative risk for total deaths and for lung cancer was higher in the matched pair study than it was when we didn't do the matched pair study.

Q Let me do it this way.

If eating fried foods corrolates with smoking, and both smoking and eating fried foods

independently correlate with lung cancer, then failure to control for either one in the study of the other would elevate the relative risks, isn't that true by definition?

A I don't think that's true by definition.

From practical example, practically, in CPS I it didn't reduce the relative risks, it increased it, and if it held up in CPS I, that was one of the reasons we didn't have to control in CPS II.

We found that controlling these factors doesn't do anything about the relative risk.

Q In what study was the matched pair study?

A It was published in 1961 in the Journal of the National Cancer Institute -- 1964; I'm sorry.

Q That was on CPS I?

A CPS I.

MR. SHEFFLER: CPS I data?

THE WITNESS: CPS I data.

MR. SHEFFLER: What that a case controlled study done?

THE WITNESS: No, it was a cohort study.

I will give you the reference if you want to wait a minute. It's not in here, my name wasn't on that paper.

If you want, I will give it to you tomorrow.

Q I would like you to give us the citation to that, but talking now about CPS II.

MR. SHEFFLER: Wait a minute, could we just have a little bit more descriptive information, Doctor?

How many people were involved in the study?

THE WITNESS: This is the study of one million men and women.

MR. SHEFFLER: How many were in the matched pairs?

THE WITNESS: This study was for men only, which would be 450,000 men, and from that we selected the never smoked and one pack a day smokers.

That reduced the number again, 80,000 nonsmokers, 70,000, and about 130,000 smokers.

So that I can't give you exact numbers, but it's in that ballpark.

MR. SHEV: You followed them for how many years?

THE WITNESS: This was followed at that time for about four years.

MR. SHEFFLER: Thank you.

Q Now, Doctor, you obtain a wealth of statistical information on these 1.2 million people in CPS II through the questionnaire?

A Yes.

Q I assume you have all of these various factors in the data stored in your computers, is that correct?

A It's not stored in the computers, it's stored on tapes.

Q If you wanted to run the tapes to determine the effect that weight and sleeping patterns and marital history and church membership and ethnicity and alcohol consumption and fat consumption and occupational exposures and socio-economic status and the rest played on lung cancer, you could do so, couldn't you?

A I can't, because I no longer have

control of the data.

Q When you had control, you could have done so?

A We could have, yes.

Q And the American Cancer Society could do so now?

A It's up to the people who are in charge of it now.

Q Considering that all of these have been found to be risk factors for lung cancer, why not determine the effect these factors could have on lung cancer as based upon the 1.2 million person study?

A The time you take to look at various things in the cancer prevention studies are valuable.

You pick things, it was my experience, and it's the leadership's experience now, you pick things that are most valuable to you.

Based on experience with CPS I, if we did exactly what you want to do, instead of being a 22 to 1 relationship, you are certainly not going to wipe out the relationship.

So if what you think may be true, and

it reduces it to 15 to 1 instead of 22 to 1, what good does it do to do a study like that?

I repeat what Wynder says, the overwhelming thing is cigarette smoking.

Anything else can't have any effect, can have very much effect at all.

Q It certainly has an effect on the people who don't smoke, doesn't it?

A The people who don't smoke have a relatively low rate of lung cancer.

We don't worry so much about people with colon cancer who have, or ovary cancer, which has about the same rate as nonsmokers for lung cancer.

It's a relative thing of what you worry about and what you don't worry about.

Q We were discussing the scientific method earlier and what separates a survey from scientific attempt to extrapolate a survey to an entire population.

Just to go back over some of these things, you said that you know the scientific method one must eliminate chance as a likely cause of result, biases as a likely chance of result, and

confounders.

A Right.

Q Even if a study is statistically significant and all known biases have been controlled for and all known confounders have been controlled for, it still may not establish a cause and effect relationship, is that correct?

A Then it becomes a matter of interpretation.

As I said before --

Q It doesn't by itself establish a cause and effect?

A Just the one study in itself doesn't establish it, no.

Q What are the scientific rules as you understand them for establishing a cause and effect relationship if there is a positive relative risk that is not shown to be biased, not known to be confounded and statistically significant?

A I think it's a matter of interpretation.

Q If a Court were to review this issue to determine whether the scientific method were applied, what standards could it use to determine

whether the scientific method were applied?

A You just mentioned the standards that are used for scientific methods.

If you are talking about interpretation and extrapolation to the population at large, that's a different thing entirely; that's outside of the scientific method.

Q So once you get to the question of interpretation of the data --

A Yes.

Q (Continuing) -- to determine whether the data show a cause and effect relationship, that's outside the scientific method?

A Right.

Q And that's based upon personal interpretation?

A According to general standards. But different people looking at the same data or the same sets of data can reach different conclusions.

Q That's because there isn't a hard and fast set of criteria that all people apply?

A On how to interpret it for the general public, yes.

Q A Court reviewing the question of

which of two experts' views to adopt to determine whether a cause and effect relationship were shown would not have a fixed set of criteria upon which it could determine which of the two experts' testimony it should base its opinion on?

A If the word expert is used correctly, that is an expert in epidemiology, both of them should reach the same interpretation of the results of the study.

Neither of them could -- is an expert, let us put it, in how to tell the public what to do about it.

That is the difference.

Q You have sat on panels where you and other epidemiologists have differed on interpretation of data?

A Probably, yes.

Q You have differed with Hirayama, haven't you?

A I don't know if we differed.

The one panel I worked on with him I was I think a little more cautious based on the results to that date than he was.

I think that's the best way to put it.

I know you hate for me to digress, but let me give you an example that may clarify this.

I recently sat on a panel in which we reviewed all the evidence on aspirin -- the use of aspirin and colon cancer, and the consensus of the panel said it is too early to draw a conclusion that we should tell the public about it.

In my personal case, I was satisfied that the evidence was sufficient to show cause and effect, and that the negative effects of taking aspirin were so minimal that in my own case, I made the interpretation in my own case I should take aspirin based on the evidence, even though the consensus in our meeting was less cautious -- was more cautious.

Q Now, one factor that you as an epidemiologist would consider in determining whether a relative risk over 1 demonstrated a cause and effect relationship would be replicability, wouldn't it?

A Definitely.

Q If studies are not replicable, then they cannot be interpreted as demonstrating any kind of cause and effect relationship, is that correct?

A I think you have to evaluate the strength of the study, how well it was done, how many cases they had, a lot of other things; not just take the P value in itself.

Q Let me address you to Exhibit number 5, previously marked.

I would like to direct your attention to Page 32 of your article on the principles of epidemiology.

A All right.

Q You see in the first column, the second paragraph?

A "The major method"?

Q Yes, the last sentence, "A finding in one study has to be replicated by other studies before a result can be accepted."

A Right.

Q You still believe that to be true, don't you?

A I certainly believe you should have replication, yes.

Q Now, another thing you need to demonstrate would be dose relationship, is that correct?

A If you have a dose response relationship, it certainly adds to the value of the relationship, yes.

Q And if there is no dose response relationship, it would tend to demonstrate there isn't a cause and effect relationship, isn't that true?

A No, that's not necessarily true.

If you take a lot of studies, all of which show an overall relationship, some show dose response, others don't, the ones that show no dose response relationship don't necessarily negate all the findings of those that do.

Q Let's take that back, let's go back on that. There are two parts of it.

One is you are saying that if every study doesn't show a dose response relationship, it may not negate the findings of the other studies, is that correct?

A That's true.

Q But if no dose response relationship is established in the literature, then a cause and effect relationship cannot be drawn, is that correct?

A It all depends on what other evidence you have, how strong the other studies are.

I can't -- hypothetically you can't make a statement like that.

Q I want to know what the standards are that a Court could use in evaluating this subject.

If not one study, but if the studies as a whole showed no dose response relationship between an exposure and a specific disease, that would tend to imply that there was no cause and effect relationship, wouldn't it?

A I would lean in that direction, but as I said, I would have to see what the studies are.

Q You, in fact, have relied upon dose response relationship to support your belief that cigarette smoking causes lung cancer, isn't that right?

A That certainly is true, and if there was one study that didn't show a cause and effect -- a dose response, I would say there is something wrong with the way the study was done.

Q But as to lung cancer in general, you have found on more than one occasion a dose response relationship between the amount smoked and the level

of lung cancer?

A Or the level of let's say precancerous findings, as we did in the Auerbach studies.

Q That goes to another question.

You have looked at dose response relationship to determine whether low tar cigarettes pose less of a health threat than high tar cigarettes, is that correct?

A Right.

Q You have found that the higher the tar delivery of the cigarette, the more likely that the individual smoker may develop cancer?

A Right.

Q Based upon that dose response relationship as you have defined it, you view low tar cigarettes as posing less of a health threat than high tar cigarettes?

A In my opinion they do pose less of a threat, yes. Not that they don't pose a threat, they pose less of a threat.

Q And you have also said that the number of cigarettes per day is correlated with lung cancer in general, is that correct?

A Right.

Q And that also is a form of dose response relationship, is that correct?

A Right.

Q And based upon that, you have said that that further supports your view that cigarette smoking causes lung cancer, is that correct?

A That's correct.

Q Now, so you have relied heavily on the dose response relationship between tar intake and lung cancer to support your view that cigarette smoking has been shown to cause lung cancer, is that correct?

A Well, we haven't relied so much on the tar intake, but on the dose relationship -- the dose response factors, number of cigarettes a day, age began smoking, depth of inhalation, quantified as best we can.

All these show a dose response relationship with lung cancer.

Q And all of those dose response relationships have been used by you in countless papers to support your contention that cigarettes have been shown to cause lung cancer in general, is that correct?

A Yes, they have shown a relationship between smoking and lung cancer, yes.

Q Even if there is a dose response relationship, and even if studies are replicable, and even if there are no known confounders or biases and the numbers are statistically significant, a cause and effect relationship still may not be shown, is that correct?

For example --

A It also depends on the strength of the relationship, the relative strength.

Q Another question is the strength of the relationship?

A Yes.

Q That's what Dr. Wynder has written on, among others?

A I assume so, I don't know.

Q Relative risks below 2 may pose difficulties in interpretation?

A Dr. Wynder has talked about that, he has written about that.

Q That relates to the strength of association?

A Right.

Q When a relative risk of 2 means that there is twice the rate of disease among the exposed than the unexposed population, correct?

A Right.

Q Epidemiology is not an exact science the way physics is, is that correct?

A Right. Physics isn't an exact science either. There are a lot of variations there, too.

Q Some causes of disease are not yet known, is that correct?

A Sure.

Q So, it's not possible to control for all confounders, since not all confounders are found, and it's not possible to control for all biases, since not all biases are known?

A In general that statement is true.

Q And because of that, when relative risks are low, which is to say below 2, they may result from uncontrolled, unknown biases or confounders, is that correct?

A Yes. There is also chance variations surrounding the risk factors.

Q And there are also random risk factors.

The lower the relative risk, the more difficult it is to interpret the results, is that correct?

A I think in general that's correct.

Q You are familiar with published literature suggesting that women who have had abortions have a relative risk of 1.5 for breast cancer?

A I saw that paper, yes.

Q But the relative risk is too low to be confidently interpreted, is that correct?

A Without a lot of replicability and supporting data, I would say that finding in itself is not enough to take public health action.

Q That is to say, a relative risk of 1.5?

A In that particular case, yes.

Q Now --

A In some cases a relative risk of 1.3 may be enough to take public health action.

Q When you say to take public health action, you have written extensively that public health action may be taken even when scientists, using the scientific method, would not believe that

a cause and effect relationship had been demonstrated, is that correct, because public health authorities must act on a lower level of information?

A I'm sorry, I didn't get that whole question.

Q Let me direct your attention to Garfinkel Exhibit number 5.

A Yes.

Q I would like to direct your attention in particular to Page 37.

Do you see Paragraph 3?

A I am not on 37 yet. Paragraph 3 on the left side?

Q Yes.

A "The risk"?

Q "Regulatory agencies."

A Okay.

Q You wrote, and I believe you have written elsewhere for the same thing, "Regulatory agencies, which are responsible for protecting the worker in the workplace from products containing suspected carcinogens and are responsible for the quality of our

air and water, have a policy of protecting the public from potential carcinogens.

"That is, their criteria for accepting study evidence on which to take action may not be the same as the consensus in the scientific community.

"Although their actions are often understandable because of their mandate to protect the public, these policies frequently create controversy and confusion."

A Right.

Q Now, when you said the public health authorities don't have enough information to take action on abortion and breast cancer, you were referring to a different level of scientific information than would be necessary for the consensus in the scientific community, is that correct?

A I would divide it two different ways. The scientific community would not accept a risk factor of 1.5 as something they would recommend to regulatory agencies without much more data confirming it.

If this has to do with a disease that is inevitably fatal, some efflux from a factory or something related to the people who live around the factory, and there is a slight increased risk, a regulatory agency might lean over backwards to say you can't do it, because even though it's not proved, we have to be overly cautious.

That's the idea I was writing about.

That applies to things like radon, for example, where they made some estimates, one of the people in the agency who made the estimate told me they have to be deliberately, I don't know if you call it conservative or not conservative.

MR. SHEFFLER: Overly cautious?

A Overly cautious, because even though the evidence isn't all in, it could be causing deaths, and they have to protect against it.

That's the attitude I am trying to get across.

Q That was the Environmental Protection Administration that you were referring to?

A I forgot what agency it was.

Q On radon? Just to clarify the question?

A This arose specifically in relation to their estimate of how many lung cancers could be caused by radon.

Q The estimate of the number of cancers that may be caused by radon was made by the EPA?

A I'm not sure what agency it was, it was a government agency.

It may have been the EPA, I'm not sure. There are some people who agree with what his estimate is. Others may not agree.

Q Mr. Garfinkel, do you recall having made a guest editorial on the environment and cancer, putting the risks into perspective, in 1990?

A My goodness, you people follow every little word I ever wrote?

Q You are an important man.

Do you recall having written such a guest editorial in CA?

A I wrote an editorial in CA. Was this in relation to the radon article that appeared?

Q Yes.

A Yes, I guess so.

Q In 1990?

A About that time, yes. What did I say

in it?

Q You said, "Scientists at the Environmental Protection Agency," EPA, "or other agencies have found it useful to make projections on the future effects of exposure."

A Yes.

Q "Based on the evidence they have now, some of which is extremely limited, but without adequate exposure data for the population of risk and with uncertain risk relationships, it is very difficult to make accurate projections for the whole country."

For example, the EPA's projection of 5,000 to 20,000 lung cancer cases per year from indoor radon exposure is based on such data?

A That was the basis for what I said in Paragraph 3.

Q So when you said --

A I should add to this there are many good scientists who agree with that estimate. It just happens I don't.

Q Notwithstanding the agency scientist who you were referring to was a scientist from the EPA?

A Yes, I guess it was.

Q And it was the EPA who said for public health purposes they are going with incomplete data?

A They didn't say that exactly. They just said we have to be overly cautious in protecting the public.

Q Where a substantial portion of the scientific community would question whether a cause and effect relationship could be shown by a weak association, that is 1 below 2?

A Yes.

Q Regulatory authorities may, for protection of the public health act, on such low or weak relative risks, is that a fair statement?

A I think a lot of it depends on how many people are exposed to it.

If a very large population, part of the population is exposed, and the relative risk as best we can determine it is less than 2, and there is replicability, and there is ancillary evidence of one sort or another, the agency probably in my opinion is justified in acting on it.

Q But the agency might be justified in acting upon that based upon an abundance of caution

for the health of the community, rather than upon a scientific standard that questions whether a relative risk of 2 can be sufficient to demonstrate a cause and effect relationship?

A I wouldn't word it that way.

I would say despite the fact that the relative risk is less than 2, there is a good possibility that even though the entire scientific community does not accept its relationship, that the relationship is real, and therefore, because so many people are involved, we ought to make some statement about it.

Q So there is a good possibility, but it has not been generally accepted by the scientific community?

A Not all members of the scientific community would accept.

Not a great preponderance of the scientific community would accept.

Look, you still found some people until very recently who denied that there was a relationship between cigarette smoking and lung cancer.

Q Members of the scientific community?

A Yes, or somebody who had an M.D. or PhD after their name.

Q Now, even if a relative risk is not weak, which is to say over 2, and even if it is replicable, and even if it's statistically significant, and even if all known biases and confounders have been eliminated, there still may be circumstances in which that relative risk does not demonstrate a cause and effect relationship; is that right?

Let me give you an example.

A All right.

Q You saw no articles in The Lancet and elsewhere that show that smokers are more likely than nonsmokers to be murdered; and the number is statistically significant and there is a dose response relationship.

That doesn't mean that smoking causes people to get murdered, does it?

A No.

Q So --

A But in that particular case, you have not eliminated all the confounders or biases, biasing factors.

Q All the known confounders and biasing factors?

A All the known confounders.

Q After eliminating for socio-economic status, which would presumably be the biggest one --

A Probably, yes.

Q (Continuing) -- the number was still statistically significant and positive.

Are you familiar with the study?

A No, I'm not.

Q Nonetheless, you don't dispute that there can be spurious positive relationships?

A There definitely could be, sure.

Q Now, how do you test to determine whether a relationship is a spurious or a true one?

A There is also the concept, which Ernst Wynder among others and I have also mentioned, and that's what we call biological plausibility.

Q What is biological plausibility?

A It has to make sense on a personal basis.

If all we had was the epidemiological evidence of relationship between cigarette smoking and cancer, you could still -- let me give you a

better example.

Just cross out what I said.

Let me give you an example. There's a lot of controversy that I guess still exists on the relationship between environmental tobacco smoke and cancer; lung cancer.

Despite the fact that there are thirty studies that have been done, the great majority of which have shown a very small increased risk, that led the EPA to be convinced that this was a carcinogen that should be controlled.

What adds great credence to the relationship is that it's biologically plausible that inhaling somebody else's smoke can effect the tracheobronchial tree in the same way that it was demonstrated that active cigarette smoke affects the tracheobronchial tree.

In addition, if you find that in checking the urine of people exposed to smoke it shows some evidence of metabolative nicotine being there, this adds to the fact that it can get into the body.

So this is what I mean by biological plausibility.

In addition to the weak epidemiological evidence, and it's weak compared to some other things, it's biologically plausible that such an effect would take place.

Q I think I understand what you are saying, and as far as ETS is concerned, we will reserve on that until we get to the Marks deposition, since ETS has been raised in that case.

For our current purposes, you are saying that a check on epidemiology is biological plausibility?

A It's not a check, it's corollary evidence, ancillary evidence.

Q In order to interpret an elevated relative risk as demonstrating or implying a cause and effect relationship, the relationship must be biologically plausible, is that correct?

A Put it the other way, if you can't really envision biological plausibility, it makes it very difficult to say that it's really a cause and effect.

Q Well, who determines biological plausibility?

A Common sense; evidence based on common

sense.

Q There are no standards?

A There is no standard that says this is biologically plausible and this isn't biologically plausible.

Q Could I determine biological plausibility as a lawyer?

A I think if you take the case of environmental tobacco smoke and I gave those two examples of biological plausibility, you would weigh that in with the other evidence.

Q But wouldn't an expert in biology or a medical doctor be necessary to determine biological plausibility?

A I don't think so; I don't think so.

MR. GROSSMAN: Let me mark this as Garfinkel Exhibit 9 for identification.

(The above described document was marked Garfinkel Exhibit 9 for identification, as of this date.)

Q Mr. Garfinkel, let me hand you a copy of what has been marked for identification purposes as Garfinkel Exhibit number 9, which is a German interview with an English translation.

I would like to direct your attention to Page 8 of the English translation.

A What page is the German translation?

Q I have no idea.

A The English translation starts on Page 13.

Is it out of order?

Q It may be out of order.

Addressing your attention to Page 8 --

A I remember this now.

Q (Continuing) -- this is a transcript of a meeting that you had in Germany addressing the subject of environmental tobacco smoke and on which you appeared on a panel with Dr. Hirayama, is that correct?

A It was a workshop, yes.

Q And Dr. Hirayama --

A And it was in Vienna, not in Germany.

Q And Dr. Hirayama reported an increased rate of suicide among nonsmoking women married to smokers, is that correct?

A I felt very sorry for my friend Takishi.

Q But that is what he reported, is that

correct?

A Well, he never reported this in a published paper, to my knowledge; he just mentioned this in the interview.

Q But his statistics showed that women married to smokers were more likely than women married to nonsmokers to commit suicide, is that correct?

A That's what he said. Now, as I say, he never reported this in a paper, to my knowledge.

Q When you say he never reported it in a paper, he never submitted it to peer review?

A It was never in any paper that was published.

Q The fact is that --

A You may find something that I am not aware of, but --

Q But he never submitted it to peer review?

A I don't think so.

Q What's the importance of submitting things to peer review?

A You have what your findings are evaluated by a competent person in the field of

expertise.

If he doesn't find it's scientifically -- scientifically valid, he will say that.

It has to be investigated or not said or whatever.

Q In the scientific community?

A Yes.

Q Is review by a peer review committee considered an important criterion for interpreting a study?

A It's an important criteria before you publish a paper for the public to see, yes.

Q So, you would not rely in general on materials that have not been submitted to peer review, is that correct?

A It depends on what you are talking about.

I mean, there are articles written that are not peer reviewed that are also very good.

It all depends what context you are talking about.

Q In the context of determining whether a risk has been established, you would not trust

findings unless they had been submitted to peer review, is that correct?

A I find it very hard to answer that question.

Q If someone reanalyzed the data concerning breast cancer from previous studies and based upon that reanalysis determined that abortion had a relative risk of 4 for breast cancer; would you rely upon that asserted relative risk if it had not been passed by peer review?

A I probably would give less credence to a paper that appeared in a journal that was not peer reviewed than one that does.

On the other hand, I have seen papers that appeared in the New England Journal of Medicine which if it was said to me as a reviewer, I would have rejected it.

Q So there are two parts, then.

Some articles that have passed by peer review are still not worth giving full credence to?

A In some people's opinions. Obviously in the editor's opinion it was worth publishing.

Q But you would still challenge certain papers that have been published after passing peer

review?

A I have in the past, yes; not many, but I have.

Q On the other hand, you could not give weight to findings that had never been submitted to peer review, is that correct?

A That isn't true. I have written articles for several journals that are not peer reviewed and I think they are pretty good.

Q You may think they are pretty good, but obviously one question that a Court has to address is whether it can accept findings.

Now, if findings have been submitted to peer review, they have been passed by the scientific community.

A Yes.

Q And if they have not been submitted to peer review, there is no independent basis upon which they can be analyzed or respected?

A I don't know how to answer that question.

I would say if you are trying to make a decision whether something is real cause and effect, and you have four papers that show a cause

and effect that were peer reviewed, and two papers that showed the same cause and effect that were not peer reviewed, I would simply say I am only basing this on four and throw out the other two.

Q Let's put it this way. What if you have two papers that have been peer reviewed that show no association, and one paper that has not been peer reviewed that shows an association?

A There is no doubt in my mind I would reject the hypothesis. I would say it's probable that there is no effect.

Q And it could not be established that there was a positive association on the basis of a single unreviewed paper in the face of two reviewed papers that showed no association, is that correct?

A Or even in the face of no papers that show no effect.

Just one paper not being reviewed certainly shouldn't be taken as something you should take public action on, public health action on.

Q Similarly, if someone made a statement in a seminar or at a meeting --

A Right.

Q (Continuing) -- that had not been

based upon peer review, that would not be the basis of action.

A Sure.

Q And you wouldn't suggest that a Court take judicial action on the basis of such a statement, would you?

A I am sure they wouldn't.

Q Getting back to Hirayama, Hirayama said in support of his findings that suicide was more common among the wives of smokers than the wives of nonsmokers, and I will quote him.

"I have a hypothesis for this phenomenon. It can be assumed that there are two different types of person, those who can easily tolerate the smoking of others and those who cannot.

"Suicide is the only means of escape for women of the latter type who cannot avoid exposure to smoke for social reasons, while women in the former category develop lung cancer."

Now I have a couple of questions relating to Dr. Hirayama's comments.

First, do you recall him having said that?

A Could I go off the record?

Q No, I want this on the record.

MR. SHEFFLER: Tell us the truth.

MR. GROSSMAN: You can go off the record.

(Discussion off the record.)

Q Back on the record.

A I guess I have to have an answer to your question.

Q I will rephrase the question.

Do you recall having heard that Dr. Hirayama had made that statement?

A I recall reading that he had made that statement in an interview.

Q In a separate interview conducted at the same time, you were interviewed?

A Yes, about the same time.

Q Now, is that what you mean by biological plausibility?

A That is not a good example of biological plausibility.

Q But is there any standard apart from just plain horse sense to determine whether something is biologically plausible?

A If there are any standards for it, I

don't know how to word it.

Q So it's in the eye of the beholder?

A No, it's based on biological evidence which lends plausibility to the hypothesis; that's the only way I can tell you.

Q Now, what biological evidence have you referred to in your published literature to support the theory that cigarette smoking causes lung cancer?

A The best biological evidence that I have referred to, the whole series of Auerbach studies which show the progression in autopsy cases of histological changes in the tracheobronchial tree in relation to the amount of smoking that they did during their lifetimes.

Q What Auerbach studies have found, and you have been a joint author on some of those studies, is that correct?

A Most of them.

Q What Auerbach studies have found in that regard is that the more one smokes and the higher tar cigarette one smokes, the more changes are found in the bronchial tree?

A Let me reword that. The more one

smokes -- the more one smokes, the greater are the amount of changes in the tracheobronchial tree.

I wouldn't put the word tar in there. That was only one study that he did.

Q In one study he found that the higher the tar delivery of the cigarette, the greater the number of changes in the tracheobronchial tree?

A Correct.

Q And also people who stopped smoking were found to have a reverse of those changes?

A Right.

Q So the changes that you are referring to are largely reversible before the onset of cancer?

A In great part, according to the Auerbach studies, the cellular changes that precede the advent of lung cancer are reduced in people who gave up smoking.

Q In fact, in an article published by you and him in the New England Journal of Medicine, you said that "Basal cell hyperplasia, a reversible change, is presumably a reaction to some deleterious factor and it is probably protective, rather than harmful"?

A I would have to read that in context, I don't know what that means.

Q The changes in the tracheobronchial tree that you referred to were largely basal cell hyperplasia?

A Basal cell hyperplasia, atypical cells, one time we called it stratification and squamous metaplasia.

All of these changes are much fewer in the lungs of autopsied ex-smokers than they are of people of the same age who are current smokers up to the time of death.

Q The appearance of basal cell hyperplasia, great many atypical cells, and the other changes that you are referring to, would be evidence that a person's cancer, if he did have lung cancer, arose from smoking?

A That's our interpretation of it, yes.

Q On the other hand, if you didn't have those changes in a person, that would be evidence that the person's cancer did not arise from smoking?

A If he had none or very few of these changes and there is still some slight evidence of basal cell hyperplasia of people who never smoked,

as they get older, or if he was an ex-smoker, the changes would be much fewer than they are in smokers.

Q If he had very few of those changes and did not have a tumor that arose in the bronchus, didn't have bronchial cancer, then his cancer could not be related in terms of biological plausibility to a smoker, is that correct?

A I don't understand that question.

If he didn't have the changes and he didn't have a bronchial tumor, bronchial cancer, then it couldn't be related to what?

Q If a person develops cancer, but not in the bronchus --

A You mean a cancer of the colon or the stomach or someplace else?

Q Or the periphery of the lung, the parenchyma of the lung?

A All right.

Q And if that person does not have cellular changes of the kind you have described in the bronchus, that would suggest that his cancer was not smoking related, is that correct?

A The few cases where cancer arises

without evidence of smoking, I would suppose that the tracheobronchial tree in those cases would show very few of these histological changes.

Q And whether or not a person smoked, if his tracheobronchial tree did not show this kind of evidence of histological change, then a cancer arising in the periphery of that individual's lung would likely not be related to smoking, is that correct?

MR. COVERT: I will object to this. I think this is out of the realm of Mr. Garfinkel, who is not a medical doctor, but an epidemiologist.

MR. GROSSMAN: It's what he's written about.

A You just said the same thing again.

Q What is your answer?

A My answer is if the person is not a smoker and if he doesn't -- if he has a tumor in his lung, a bronchial tumor in his lung or in the periphery of the lung outside of the bronchus, the chances are there would be very little changes in the tracheobronchial tree.

Q That's not what I am asking.

I am saying if it were determined on

bronchoscopy or autopsy that the person's lungs had not undergone the changes that you have described in your published articles --

A Yes.

Q (Continuing) -- but that person had nonetheless had a smoking history, and that person had a tumor in the periphery of his lung, the actual pathological finding that he did not have changes in the bronchus would tend to suggest that his cancer did not arise from smoking, is that correct?

A I misunderstood what you said.

You are talking now about a smoker?

Q Yes.

A I say that your hypothesis can't be true, because every smoker we had in our studies, which was easily 2,000 cases, easily, and we looked at fifty-five slides for each lung, so there were thousands and thousands of slides.

In everybody who reported a history of smoking we found some histological changes.

It was less in those who smoked minimally than those who smoked more, but we -- there never was a person -- I don't know if I should say never.

If there was a person, I would have remembered it, and if it was, it would have to be somebody who was very young.

But anybody who smoked had far more histological changes than any of our never smoked.

So I think your hypothesis that there was no changes in these people is untenable.

Q It is a hypothesis, and since it's hypothetical, and since you have been offered as an expert, I just ask that you answer in terms of the hypothesis.

And I understand as someone who is not a pathologist you are not in a position yourself to look at the histological changes and make a diagnosis.

But within that context, if a person reported a history of smoking, but nonetheless was found by a pathologist not to have the kinds of changes that you have reported in your literature as being precursors of cancer, wouldn't that suggest that cancer arising in the periphery of that person's lung was related to causes other than smoking?

A I really can't accept your hypothesis,

it's impossible.

Q You have to accept the hypothesis.

A If I accept this very unlikely hypothesis, then I will agree with the statement.

Q There is no general acceptance in the scientific community of a specific mechanism of lung cancer, is that correct?

A The general consensus in the scientific community is that there is something in the tar element of tobacco, suspended particles, that is the putative cause of lung cancer.

Q I am asking about mechanism.

A Mechanism, how does it start?

Q Yes.

A From the Auerbach studies, I would say that there is a whole spectrum of changes, and we use this word in one of the articles, that starts with basal cell hyperplasia, some of the cells become atypical, the cilia starts to exfoliate, eventually it develops into what we call carcinoma in situ, which is a whole area of atypical cells.

And that in some of these instances, and we don't know exactly how, the cells break through the basal membrane and become a full fledged

cancer.

Some of these areas are carcinoma, develop into lung cancer, in others would stay as carcinoma in situ for long periods of time until death, we don't know.

There are many, many more areas of carcinoma in situ than there are of early cancers.

But the mechanism is that we never find a lung cancer without it being embedded in an area of carcinoma in situ.

Q And it's your theory taken from Dr. Auerbach that the carcinoma in situ is a developmental stage along the road that begins with those basal cell metaplasias and hyperplasias?

A Yes.

MR. SHEFFLER: Is this a good place to take a break?

MR. GROSSMAN: I think it is.

(Whereupon, at this point in the proceedings there was a recess, after which the deposition continued as follows:)

MR. GROSSMAN: Let's go back on the record.

During the break we discussed timing,

which has become a difficult problem because depositions were noticed for Mr. Garfinkel in three separate cases and a videotape was noticed in two cases of Mr. Garfinkel.

No one has tried to stop Mr. Garfinkel from giving extended answers and we all recognize this is a long process.

What we have agreed is that we will continue the Gilboy deposition until 5:00 today, at which time it will not be completed; that is, Jerry, we understand that the discovery --

MR. COVERT: It may not be.

MR. GROSSMAN: (Continuing) -- the discovery deposition in Gilboy will in all likelihood not be done at 5:00, and you understand that?

MR. COVERT: Right.

MR. GROSSMAN: We will not have, it's been agreed, a videotape deposition in the Gilboy case tomorrow.

MR. COVERT: Let's don't make that agreement, Ted. If miracles happen and we complete both of them tomorrow, we have the video here; it's been noticed, so we can just go from one to the

other.

I don't see how that's a possibility, but I'm not going to stipulate myself out of this possibility happening.

In other words, if you finish at 9:30 in the morning, 10:00, and Bruce finishes in three hours at 1:00, and both depositions are finished at 3:00, when we start his, we will just go into yours afterwards; if we haven't finished we haven't finished.

MR. GROSSMAN: We will take care of tomorrow for tomorrow, but when we have talked, Jerry, you have said that you understand that it's at least highly likely that we will not get to a Gilboy videotape tomorrow.

MR. COVERT: Just play it by ear is what I am saying.

If it does happen, it happens; if it doesn't happen, we are going to concentrate on Arabie.

MR. GROSSMAN: In any event, for now we will go until 5:00 and we will then break.

MR. COVERT: 9:30 tomorrow.

Q Now, Mr. Garfinkel, when we took our

break we were talking about biological plausibility.

I am trying to determine for the record what standards exist to determine biological plausibility.

Is it a fair summary to say that there are no written standards for what determines biological plausibility?

A Not to my knowledge.

Q Is it a fair statement to say that different people could reasonably disagree as to what is biologically plausible?

A That's true.

Q Is it fair to say that there is no written standard of any kind by which you could determine which of two people was right in saying that a relationship was or was not biologically plausible?

A I don't know of any standard.

Q Now, let's turn quickly to the definition of terms.

What's the difference between relative risk and odds ratio?

A Odds ratio is abbreviated, it's estimated odds ratio theoretically.

In a sense they are the same thing, it's the risk of developing a disease of an experimental group, exposed group, compared to a control group.

Q Now, a relative risk measures increased risk from exposure over the risk in the population as a whole, is that correct?

A That's not said correctly. It's the risk of exposure divided by the control group which stands for the population as a whole.

Q And the control group presumably has no known exposures to --

A No, the control group does not have the exposure that the experimental group has.

Q Well, if you were doing a study for ETS, the relationship between ETS and lung cancer, if the control group were active smokers, would that skew the numbers?

A If you are doing a study of ETS, you want to compare -- the way it is generally done, you compare let us say nonsmokers who are exposed and who get lung cancer versus nonsmokers who are exposed who get another disease or don't get -- or don't get any disease at all.

So, it's a little different than the context of which you are talking about.

Q So confounders are controlled for?

A And presumably confounders are controlled for in this particular case.

Q If confounders are controlled for, then the relative risk should measure only the difference in incidence applicable to the exposure, is that correct?

A Theoretically, if you are aware of all the major confounders, or ones that could make a difference.

Q If the confounders are not controlled, the study may be flawed?

A It may be flawed, yes.

Q Now, sometimes -- you said earlier that epidemiology is not personal but rather it's based on the population as a whole?

A Based on population groups.

Q It doesn't tell you the cause of any individual's disease but rather describes the incidence of disease among the exposed and unexposed groups of the population?

MR. COVERT: I will object to that

Ted, I think it's been asked and answered.

MR. GROSSMAN: I am just saying -- it has been answered and I am just saying that as a basis for the next question.

Q Mr. Garfinkel, some exposures are believed to be synergistic with others and some are believed to be independent, is that correct?

A That's true.

Q Now, there is published literature suggesting that asbestos and perhaps radiation exposure may have a synergistic effect with tobacco exposure for lung cancer, is that correct?

There is such published literature?

A I am not familiar with that literature, but I am familiar with the literature that asbestos and cigarette smoking are synergistic and add considerably to the risk.

Q That is, that the two exposures taken together may produce a level of risk higher than both of them added together?

A Yes, it's generally called multiplicative risk rather than an additive risk, when they are independent.

Q And there is literature to that

effect?

A That's right.

Q There is also a belief that other risks, other types of exposure, may be additive with cigarettes in determining risks in the population for lung cancer, is that correct?

A That's right.

Q For example, diet would be an additive risk, is that correct?

A I am not aware of a paper that showed that, but it certainly could be true; I don't know.

Q There is a presumption that risks are additive unless otherwise shown, isn't there?

A I don't know if a priority can establish whether a risk is additive or multiplicative. I just don't know.

Q It's generally believed that alcohol is an additive risk, is it not?

A Alcohol and tobacco are probably additive risks for let us say cancer of the larynx.

I think they would probably be additive, although I don't know of any study that looked at it together.

Q Exercise is a risk, lack of exercise

is a risk --

A We did a study on alcohol and mouth cancer. I would say alcohol and cigarette smoking are multiplicative risks for mouth cancer.

Q But for lung cancer?

A Lung cancer, I don't know who has looked at it, I don't know. There may have been somebody, I just don't know.

Q How about lack of exercise, that's a risk factor for cancer, isn't it?

A Lack of exercise has been reported to be related to colon cancer, and some other cancers.

Q Family history?

A Family history is certainly related to breast cancer and a few other cancers; retinoblastoma seems to have a heredity input, and there are certain syndromes, Li-Fraumeni syndrome, which relates certain cancers to family history.

Q Are you family with a fellow named Jonathan Samet?

A Oh, yes.

Q He's a Chairman of the epidemiology department at Hopkins?

A Yes.

Q He has published reports indicating that individuals whose parents had lung cancer had a relative risk of 5 for lung cancer after controlling for cigarette smoking.

Are you familiar with that?

A I am not familiar with that paper at all.

Q Are you familiar with the Surgeon General's report of 1989, reducing the health consequences of smoking?

A I probably read every page of that report.

Q I don't have a copy of it with me, although -- we have one.

Could we mark this as Garfinkel Exhibit 10.

(The above described document was marked Garfinkel Exhibit 10 for identification, as of this date.)

Q Mr. Garfinkel let me hand you what has been marked for identification purposes as Garfinkel Exhibit 10, which is some pages from the 1989 Surgeon General's report.

I would like to direct your attention

to the top of the Page 52.

It cites to a recent case control study in New Mexico, Sanborn, Helmut and Pantick, showing that a parental history of lung cancer was associated with a five-fold increase in lung cancer risk after adjusting for cigarette smoke.

A Right above that it also says "subsequent epidemiologic studies have provided empirical evidence of a possible genetic determination."

I am not familiar with that paper.

Q This doesn't refresh your recollection of having read about it in the Surgeon General's report?

A No, I don't remember reading about this.

Q Other reports are indicated also of Brisman, Lynch and Goffman showing clinical studies also indicated family aggregation?

A I know some of Lynch's work, but not all of it. I also would like to put in the record the last sentence of that section.

It says, "However, not all subsequent studies have been confirmatory and the inheritance

of inducibility in human has not yet been fully described."

Q That's on mechanism, isn't it; that paragraph is dealing with mechanism, isn't it, sir?

A Well, it says, "This observation suggests possible genetic determinant of lung cancer risk. Studies have not been confirmatory."

Q That's about the inducibility of aryl hydrocarbon hydroxylase?

A The last sentence in the penultimate sentence is, "This observation suggested a possible genetic determinant. Not all subsequent studies have been confirmatory."

Q Just for a clarification of the record, the first paragraph of this refers to epidemiological studies, is that correct, the Samet study, the Brisman, Lynch and Goffman studies?

A The Brisman, Lynch and Goffman are clinical studies; I don't know if they are epidemiological studies.

Q The Samet study is epidemiological?

A Samet is probably epidemiological.

Q It's a case control study?

A Yes.

Q Now, when you said that you wanted to add something to the record and you read a sentence from the next paragraph, that next paragraph is -- refers to studies on mechanism and specifically to observations of a higher degree of inducibility of aryl hydrocarbon hydroxylase which converts polycyclic aromatic hydrocarbons to more active carcinogens.

It is that which has not been confirmed by other studies, isn't that correct?

A I was just reading that statement, that the observation suggests that -- a possible genetic determinant.

Q And the observation they are talking about is the inducibility --

MR. COVERT: Ted, I object.

MR. GROSSMAN: Mr. Garfinkel decided to volunteer an additional statement into the record, so I need to clarify the record.

MR. COVERT: You have done it three times and he said he thinks it refers to the whole section.

You said it's one and I don't think you can get any more discovery.

Q Mr. Garfinkel, the observation referred to in what you read into the record is the observation regarding chemical analysis of difference between people --

A That is your interpretation of it, I read it the other way.

Q All right, Mr. Garfinkel, let's continue.

Do you know of any literature on whether family history is additive or multiplicative?

A I am not familiar with any of the literature of any of the family history being additive or multiplicative.

Q Are you familiar with any literature on whether area of residence, relative risks are additive or multiplicative with cigarette risks?

A No, I am not familiar with any studies of that sort. No, I shouldn't say that.

One of the studies that we did took residence into consideration, and as far as I know didn't show any additive risk at all.

Q You are saying that it wasn't found to be a risk?

A It wasn't found to be a risk.

Q That was in one study?

A In one study.

Q All right, doctor beginning in the 1960's, with the Surgeon General's report of 1964, there has been a marked decline in male cigarette smoking in the United States, is that correct?

A Yes, these are studies done by the National Center For Health Statistics, I believe.

The one that was highlighted in the 1989 Surgeon General's report, I think, started in 1965.

Since then there has been a steady drop in cigarette consumption per capita, yes.

Q What has been the extent of the drop?

A In round figures, in 1965, forty-two percent of males and about thirty-three percent of females smoked.

In 1992, which I think is the last year, it was approximately twenty-six percent in males and twenty-three percent in females.

Q What was the lung cancer rate among men in the United States in 1970?

A I don't have those figures offhand.

It was certainly much lower than it was in 1980 or 1990. It was a steady rising pattern in the lung cancer rate starting in about 1940.

Q Has it gone down in the last ten years?

A The incidence rate, according to the SEER Program, decreased starting in about 1986 through 1991, which is the last -- 1992, I guess, which is the last I have seen it, and the mortality rate has leveled off in the same period of time, but it has certainly decreased in those who are under 65.

Q What has been the level of decrease?

A It was greater in the younger people than in the middle aged people, but -- I can picture the graph but I can't tell you what the numbers are.

In the 65 to 74 it sort of leveled off and the 75 and over it still was increasing.

Q You and others had predicted an earlier and greater decline in the level of lung cancer, hadn't you?

A I don't think we predicted an earlier decline. I don't know if anybody -- we predicted a decline, but I don't know when we said it would

start.

It certainly has been going down, the incidence, at least of lung cancer, has been going down, fortunately.

Q What accounts for the mortality of lung cancer not going down faster given the decline in smoking that began in the 60's?

A I have to say that it's very hard to know what's happening when the situation is so dynamic.

First of all, we know when people give up smoking, the rate doesn't go down immediately, it takes some time for the rate to decrease.

I think the increased rate in those who were born after 1930 is because fewer people took up smoking in those age cohorts and it keeps decreasing.

The fact that this is related to a mortality drop in the younger people rather than the older people reflects the fact that the older people have smoked for a longer period of time and that it takes longer, I suppose, for the facts of incidence and mortality of lung cancer to catch up.

But it's a very dynamic situation, it

works different in different age groups, so it's very hard to say.

Q Let's break it down into some component parts.

First of all, because lung cancer is so often fatal, lung cancer mortality statistics are often viewed as more accurate than lung cancer incidence statistics, isn't that correct?

A Well, if you use the lung cancer incidence statistics from the SEER Program, I don't think many people would say that these are in error, or if they are in error, they can't be very much in error.

Q What would account for a declining incidence rate with a flat mortality rate?

A I think it's a matter of lag in time, as I said.

It's a dynamic situation where the forces are working differently in the younger people and in the older people and it takes some time for it to catch up.

I fully expect -- and again, it's a prediction, but I am pretty good at it -- I predicted a drop in breast cancer mortality and a

year after I said it the head of the National Cancer Institute said that there was a five percent drop, the early indications -- and the figures aren't out yet -- is that the lung cancer mortality rate, which has been pretty flat for the last five or six years, showed a drop in 1992.

I saw this in one chart, I haven't been able to verify it yet, but I expect that the continuation of the drop in the younger people will continue, the 65 to 74 will probably start to go down and the 75 to 84 group will start to level off.

So there is different forces working on each age group that may help to explain the discrepancy.

For example, the increase in breast cancer rate from 1980 to 1987, thirty-two percent increase.

It was not followed immediately by a breast cancer mortality rate. You expect it to go up, but it didn't, it stayed level, and as a matter of fact, with a lag it's going down.

Q If I can understand this, as far as the level of incidence versus the level of mortality, let's focus on that first, if incidence

is decreasing, but mortality is remaining flat, that would mean that the survival rate of lung cancer patients is declining, is that correct?

A It's affecting different groups of people, so -- it's very hard to say that.

Q Well, if fewer people are getting lung cancer, but the same number of people are dying of it, that would have to mean --

A You can't look at it in the same period of time because there is always a lag between incidence and death, even among lung cancer cases

Q How long is the normal survival time?

A If you want to look at the survival, you look at it directly from the SEER Program, they publish figures on survival of lung cancer and survival of lung cancer has gone up slightly.

Q If survival of lung cancer is going up and incidence is going down --

A I say it's going up, it went from fourteen percent to sixteen percent, something of that order of magnitude.

Q Let me finish the question.

If survival from lung cancer is going up and incidence is going down, how can mortality be

flat?

A As I said, there is a different dynamic in different age groups in the population and I think that would account for the summary figures for mortality and incidence.

Q Could you explain how a dynamic in different age groups could account for that, I don't understand?

A I don't know if I could explain it to you. It might be -- no, I won't even try to explain it, but I think that could possibly account for the phenomenom you have mentioned.

Q Is it fair to say you had predicted in print as early as the 1970's that lung cancer mortality rates would soon go down because of the steep decline in smoking in the United States?

A I probably said that in print, yes.

Q What would account for the failure of mortality rates to go down, it's now twenty years later?

A A lag in -- remember, in 1970, mortality was still going up at a fairly steep rate. I think the fact that it leveled off between 1987, let's say, and 1991 is a decrease in

mortality; in fact, a change from going up to leveling off is a decrease.

Q There has also been a change in the types of lung cancers that have been reported, isn't that correct?

A There has been an increase in the number of adenocarcinomas in that period of time, yes.

Q An increase in the period of --

A And a decrease in squamous cell

Q A decrease in squamous. There has been an increase in peripheral lung cancers and a decrease in central lung cancers, isn't that correct?

A Probably, yes.

Q There has been an increase in the number of reported scar cancers, is that correct?

A Slight increase, not much.

Q Now, scar cancers, according to the reported literature, can arise from scars formed by a number of diseases and other processes, is that correct?

A I don't know if it could arise from this.

Dr. Auerbach has looked at these more than anybody else, and I think what he says in his papers is that it's associated with such things as infarcts and tuberculous lesions and maybe some other things.

These things don't cause it, it arises around the scar.

Q Doctor, you are not an expert?

A I am not an expert in the pathology of this.

Q You would not be competent to render an opinion as to what causes scar cancer, is that correct?

A I don't think a pathologist is competent to comment on what causes it either.

He may be able to describe it, but cause and effect is something else entirely.

Q Insofar --

A He can give an interpretation, but not necessarily the cause.

Q Insofar as you are concerned, you are not competent to render an opinion as to what causes scar cancer, is that correct?

MR. COVERT: Excuse me, I am going to

have an objection, Ted, I make the same objection.

MR. GROSSMAN: How can you object to that?

MR. COVERT: I think he can render opinions because you said he could render an opinion on something that he's written on.

So if he's written on this, I think he's capable of rendering an opinion.

MR. GROSSMAN: I am asking him if he has -- I think you are trying to lead the witness here on an area that's obviously critical.

MR. COVERT: I made the same objection, the record will speak for itself.

If you want to answer the question, Mr. Garfinkel, go ahead.

MR. SHEFFLER: I would be willing to stipulate Mr. Garfinkel lacks the competence to answer the question, if you will, too.

MR. GROSSMAN: I will, too.

MR. SHEFFLER: Okay, I will take the silence as a negative, please answer the question.

Q Mr. Garfinkel?

A Yes.

MR. COVERT: What's the question?

MR. GROSSMAN: Would you read the question.

(The question requested was read back by the reporter.)

A I could give an opinion, ~~I don't know if somebody would -- I'm sorry,~~ I could give an opinion based on my association with Dr. Auerbach and my evaluation of the number of scars that appear.

~~I don't think anybody at this point knows what -- whether or not a scar formed --~~ I don't think at this point anybody knows whether a lung cancer formed around a scar is caused by any factor.

All we can say at this point, and Dr. Auerbach says it in his paper, is that ~~a~~ scar was associated with either an infarct or tuberculosis or an unknown cause.

Q Your answer contains a number of components.

First, you say that you are not sure that anyone can render an opinion as to what causes scar cancer, is that correct?

A I don't think anybody could answer

that question with any degree of certainty.

Q Second, to the extent that you have an opinion on the causes of scar cancer, it's not an independent expert opinion but is based primarily upon what Dr. Auerbach has told you, is that correct?

A It's dependent on what Dr. Auerbach described in the paper we wrote together.

Q That is because you did not conduct any pathology yourself or review the pathology yourself?

A Conducting a pathology yourself or reviewing the pathology yourself does not make you an expert on cause and effect of lung cancers and scars.

Q Nor could you render an expert opinion on the mechanism of scar cancer, is that correct?

A That's what I said. We don't know the mechanism of scar cancers, and -- Dr. Auerbach doesn't know it and I don't know it.

Q Now, one possible explanation for the increase of scar cancers that has been noted over recent years is that a greater proportion of the people dying and being autopsied survived

tuberculosis and other inflammatory diseases that would have killed them in earlier years, is that correct?

A I think some people have given that opinion of the reason why a tuberculous lesion occurs associated with a scar.

MR. SHEFFLER: Tuberculous lesions associated with a scar?

THE WITNESS: Associated with a scar, yes.

Q Let me see if I understand your answer.

A Let me read this and it will give you an exact opinion.

Q What are you reading?

A From the paper on "Scar Cancer of the Lung."

Q Which one are you referring to?

A "Scar Cancer of the Lung," Auerbach Garfinkel and Parks.

Q 1979 Cancer magazine?

A 1979.

Q Why don't we -- we have a copy of that one, why don't we mark a copy of that and place it

in the deposition record.

This is number 11?

A May I read this?

(The above described document was marked Garfinkel Exhibit 11 for identification, as of this date.)

Q Mr. Garfinkel, let me hand you what's been marked for identification purposes as Garfinkel Exhibit number 11.

That's a copy of your article with Drs. Auerbach and Parks, 1979, on scar cancer of the lung, is that correct?

A That's right.

Do you want to ask me something about this.

Q You just wanted to read something from this?

A I am just going to say on Page 637, second column to the right, lung cancer was found in so many cases they reviewed the "Lung cancer was found in 1,186 cases, all histologic sections prepared from the lung in these cases were reviewed to determine the presence or absence of a scar associated with the tumor," not causing the tumor,

"the etiology of the scar," whether it was tuberculous, or infarct and other findings of significance.

Then he goes on to say, "If caused by an infarct, former structure of the lung parenchyma may be still be apparent within the scar, if caused by tuberculosis, a central zone of necrotic tissue with calcium present."

And this is the scar and it's associated with a cancer but it doesn't say that the infarct or the tuberculosis causes the cancer.

Q It's fair to say that you didn't -- let me start that sentence again.

It's fair to say that your article and your study reflected in your 1979 article tried to determine the reasons for the increase in scar cancer over the years and tried to describe the nature and etiology as best could be of scar cancer?

A Yes, we looked at the trends of scar cancer, we looked at where they occur, we looked at the histological types associated with the various tumors we saw.

And we looked at the kinds of things that were associated with the scar, specifically

associated with the scar.

Q What things were associated with the scar?

A Infarct, fifty-six percent, tuberculosis, twenty-three percent, granulomata, one percent, asbestosis, one percent and etiology not evident, eighteen percent.

Q That's of the scar itself, is that correct?

A Yes.

Q Now, is tuberculosis associated with a scar or did tuberculosis cause those scars?

A I think the scar is evidence of a healed tuberculosis lesion.

Q That's a way of saying the tuberculosis caused those scars, is that correct?

A Yes, I think you could say that.

Q Although you used the term associated with a scar, elsewhere in the same article you referred to scars caused by infarcts and lesions, is that correct?

A I am not sure of that.

Q Let's turn to Page 641 of your article.

Let me direct your attention as an example to the last full paragraph, "In our study, that paragraph?

A Yes -- 641. "In our study" --

Q In our study. If you look down around five or six lines, "Because of the large number of scars caused by infarcts and the greater tendency for these to occur in the lower lobes" --

A Well, the scar -- the infarct and the tuberculosis lesion, tuberculosis causes the scar. It doesn't mean that it causes the cancer.

Q So, you are saying?

A These things are definitely caused by infarcts and tuberculosis lesions and asbestosis and whatever. But the lung cancer, the word we use is associated with the scar.

Q Just to clarify your testimony, you are saying that it was your understanding that the scars in question were caused in some cases by tuberculosis and other cases by other inflammatory diseases?

A By infarcts.

Q And in other cases in rare cases by asbestos, is that correct?

A Yes.

Q You also described the literature on scar cancer and you noted that Friedrich and others from the late 30's and '40s had noted the scar cancer as a separate independent type of lung cancer?

A I am not familiar with their papers.

Q Let me direct your attention to --

A "Discussion Friedrich and" -- they use the word associated, too.

Q On Page 640 you say, "Friedrich and Rossle were the first to report a series of pulmonary carcinomas which were found closely associated with previously formed lung scars and established 'scar cancer' as a separate entity among lung tumors."

Is that correct?

A That's what they said.

Q Well, the "they" is you, is that correct?

A This is Friedrich and Rossle that said that.

Q You are reporting on what they said?

A Reporting on what they said, yes.

Q Going down further in your article you cite to 1954, a Luders and Themel article reporting on over 2,000 autopsies.

A Yes.

Q And your article reports on several others describing the distinct phenomenon of scar cancer, is that correct?

A It's reported in the literature on scar cancer, yes.

Q Now, you looked in your study to determine what was associated with scar cancer, is that correct?

A That's right.

Q What types of histology the scar cancer represented?

A That's right.

Q You found that the most common histology of scar cancer was adenocarcinoma?

A The most common histological type associated with a cancer around a scar which is called scar cancer, was adenocarcinoma.

Q You also found that scar cancers were most common in the periphery, rather than center of the lung?

A Oh, yes.

Q And you also found that scar cancers were most common in the upper lobes, although they could be in the lower lobes as well?

A That's what we found, yes.

Q You found that there was no relationship between scar cancer and smoking history?

A We really didn't make an accurate analysis of that, because we didn't have a base population to look for that.

We just reported the percent of scars that occurred in smokers and nonsmokers and by amount of smoking.

Q Let's turn to Page 636. Do you see the summary of your article?

A Yes.

Q Could you read?

A The last sentence says, "No relationship was found between smoking habits and scar cancer."

I don't think that is an accurate analysis, that's the end of the quote.

Q That was subjected to peer review?

A This appeared in the Journal of Cancer and I assume it went through peer review.

Q Cancer is a peer review journal?

A Yes, it is.

Q Who wrote the sentence, "No relationship was found between smoking habits and scar cancer"?

A I don't recall who wrote that. In retrospect, I think it was not a proper thing to say, because we didn't really make an analysis, a proper analysis of smoking habits and cancers associated with the scar.

Q Who do you believe wrote that sentence?

A I mentioned that I can't recall who wrote that.

Q Let's turn to Page 639. Look at the bottom of the left-hand column.

Do you see the paragraph beginning "In 116 of the 183 peripheral lung cancer cases a smoking and occupational history was obtained."

A Right.

Q "One of these was a 46 year old man who never smoked regularly, in that case a large

cell undifferentiated carcinoma developed around a scar caused by an infarct."

A Right.

Q "Among the smokers the percent of peripheral lung cancer cases with a scar showed no increase with the amount of cigarette smoking."

A There was no dose response according to the number of cigarettes smoked per day.

Q There was no dose response whatsoever?

A There is a slight dose response, it's higher in those who smoked two packs a day than those who smoked less than one pack.

Q And it is lower among those who smoked one pack a day than those who smoked less than one pack?

A It's the same, 46 or 45 is the same.

Q Your interpretation in 1979 of this data was that there was no dose response from cigarette smoking to scar cancers, is that correct?

A My interpretation then is that there was not -- no dose response, that's correct.

I also would point out only one of these people was a never smoked; the others were all smokers.

Q If you could just limit yourself to the question.

A Well, you are giving me an incomplete question that I can't answer accurately.

Q Mr. Garfinkel, it's accurate to say that that sentence passed peer review?

A As far as I know, none of the people who reviewed the papers sent this back to us and asked us to explain it a little further.

Q Now, Mr. Garfinkel, there is a wealth of other literature on scar cancers showing no association between smoking and scar cancer, is that correct?

A I am not sure of the other papers, no.

Q Have you reviewed the other papers?

A I am not aware of any of the other papers that brought this up.

Q Could you turn to Page 640. Could you look at the second paragraph and read that into the record, please?

A The one that starts "Smoking and occupational history"?

Q Yes.

A "Smoking and occupational histories

were available for 830 of the 1186 lung cancers in the twenty-one year period and separate analyses were made of the proportions with scars by amount of smoking, broad occupational group and age at death.

"As in the analysis of peripheral lung cancer, no increase of cigarette smoking was observed.

"Scar cancer was found in 6.5 percent of the less than one pack, 7.0 percent of the one to two pack and 6.6 percent of the two plus pack.

Q Just to clarify the record, you said no increase of cigarette smoking was observed, that's no increase with cigarette smoking was observed, is that correct?

A Right.

Q Those numbers indicated to you based upon 830 cases during a twenty-one year period that there was no relationship between smoking and scar cancer, is that correct?

A Well, of course, scar cancer was only -- what we called scar cancer was only a small portion of the 830 cases.

There were 87 or so, 82.

Q Those 82 were -- constituted something

under half of the total cases of peripheral lung cancer?

A I have to check that.

Q Look at your next paragraph. Among the 183 peripheral lung?

A There were 183 peripheral cancers and 82 with scars, it's a little less than half.

Q In both the peripheral cases as a whole and the scar cancer cases in particular, you found no association between smoking and carcinoma, is that correct?

A That is not correct.

Only one of the people who had a scar was a never smoked, all the others were smokers.

So you can't really say there was no association, there was no dose response association, but you really can't say there was no association.

Q You said there was no association, didn't you?

A No increase with cigarette smoking was observed.

It doesn't say that there is no relationship between smoking to this paragraph, it doesn't say no relationship between cigarette

smoking and scar cancer.

Q In the synopsis on the first page, it says exactly what you said it doesn't, does it?

A I am saying I am not sure who wrote that sentence.

Q It could have been you?

A It's possible it was me, but I would doubt it.

Q It could have been Oscar Auerbach?

A Could have been Oscar, could have been Verda Parks.

Q It was one of the three of you?

A Right.

Q And it passed by you before publication?

A It did.

Q If you didn't write it, you approved it, is that correct?

A I didn't make any comment to delete it is --

Q You either wrote it or allowed it to be published, is that correct?

A That probably is correct, yes.

Q Now, Mr. Garfinkel, in addition to

finding no association between smoking history and scar cancer --

A Excuse me, that is not true.

There is a relationship between smoking and scar cancer.

There is no dose response relationship, and that's what I said in the inside paragraph.

Q Could you point out any place in this article, and I can wait for you to read the whole thing if necessary, where you stated that there was an association between smoking and scar cancer?

A No, the only way one could infer that is from the sentence which says, "One of these is a 46 year old man who never smoked regularly."

Q Just to clarify this record, your article specifically says that there is no relationship.

It specifically says that there is no association.

Are you taking the position that the article implies that there is no association -- that there is an association?

A I am saying that the article says

specifically there was one person who was a never smoked and that among the cigarette smokers with what we described as scar cancer, there was no dose response relationship.

I am further testifying that in order to know whether a relationship exists between those people classified as a scar cancer or -- those people classified as scar cancer and cigarette smoking, one really has to look at rates.

Q Rates of what?

A Rates of occurrence of scar cancer in people who never smoked and in people who were cigarette smokers.

Q What is the most common form of cancer among people who have never smoked?

A The most common form of cancer among people who never smoked are broadly classified adenocarcinomas.

Q What's the most common location of lung cancer among people who have never smoked?

A Among people who never smoked it is more likely that the cancer occurs in the periphery than in the proximal part of the lung.

Q How many peripheral adenocarcinomas

are there among smokers every year in the United States?

A I don't know if anybody knows that.

Probably a study of the incidence data would be -- would have to be used to get an estimate of the number who died.

I don't have that offhand.

Q You testified earlier today that there are 15,000 people a year or so who never have smoked and who develop lung cancer?

A Right.

Q And who die of lung cancer every year?

A Who die of lung cancer every year.

Q 15,000 nonsmokers, lifetime nonsmokers every year die of lung cancer?

A All right.

Q And the most common kind of lung cancer among those people is peripheral adenocarcinoma, is that correct?

A The most common type is peripheral adenocarcinoma.

We can't say what percentage is from this paper, because virtually every one of these people smoked.

Q What percentage of individuals who smoke develop peripheral adenocarcinoma?

A I have no -- well, if you use this data -- and, of course, it could have changed, these were data collected from 1955 to 1976 -- of all the lung cancers -- and these are all smokers -- by and large, fifteen percent develop peripheral cancers.

Q Peripheral adenocarcinoma?

A ~~Peripheral cancers of the --~~ If you just look at peripheral adenocarcinomas, that proportion would be much less.

There ~~is~~ are some squamous cells and some large cells that develop and some bronchioloalveolar cells develop in the periphery.

Q What I am asking is, so far as you know in the population as a whole, what percentage of smokers who develop lung cancer develop peripheral adenocarcinoma, in the population as a whole?

A Well, I have no idea in the population as a whole.

If we use this data to make the estimate, it will give you a ballpark; unless it's increased tremendously, it's 59 out of 1186 total

lung cancers.

So that would be about six or seven percent.

Q Six or seven percent?

A But I'm not sure if this is an accurate estimate of what it is today.

Q Let's just see how that works out.

What percentage of the total population as of 1979 was ever smokers?

A I will have to withdraw that statement.

It's 93 -- 95 peripheral adenocarcinomas out of 1186.

How much is that?

Q Eight percent?

A Roughly eight percent.

Q What percentage of the adult population in the period of the 1970's was ever smokers?

A In 1970, you say?

Q Yes -- 1960's and 70's?

A I would say somewhere around sixty-five percent would be ever smokers.

Q Of the 130,000?

A We are talking about men now; women -- if you do women, it would be less.

Q When you testified earlier that there are 130,000 or so lung cancer cases a year among smokers, you are including ever smokers or current smokers?

A I think that figure refers to ever smokers. This is 1994, but it's close enough.

Q Let's just do a quick analysis. If --

A Excuse me, let me clarify the figures that I gave before.

In 1994 we estimate that there was 172,000 lung cancers.

Q Incidence or mortality?

A Number of new cases, of which 100,000 were male and 72,000 were female.

Now, I imagine 1995 data, which I don't have, it's about the same.

There are 153,000 deaths, 94,000 are male, 59,000 female.

Q You also testified that you understood that there was about 130,000 deaths from lung cancer among smokers, and I assume -- and it's your testimony that that was ever smokers?

A Our latest current estimate from the Cancer Society is about ninety percent. Let me just verify that. Eighty-seven percent of lung cancers we attribute to smoking.

So if you take eighty-seven percent of the 153,000 deaths, that would be how many we attribute to smoking.

I don't have my calculator. If you take -- 50,000 would be ten, take off another 3,000, 18, so it's about 135, about 135,000; I said 130, about that.

Q How many deaths in total did you say?

A Now, this is last year's estimate, 153,000.

Q Just so we are clear on this, you testified earlier that you thought that there were 15,000 deaths among lifetime nonsmokers?

A That's what general estimates are.

Q In fact, using the estimates that you have just given, there would be 20,000 deaths?

A I said 138, so that would be about 15,000.

Q The percentage that you have, which is thirteen percent, I believe?

A Yes, thirteen percent of 153, how much does that amount to?

Q 19,890.

A All right.

Q So the correct number would be 20,000?

A Roughly 20,000, okay.

Q Not 15,000?

A Right. I have seen some estimates of 15,000; some people say ~~it's~~ ninety percent rather than eighty-seven percent, so that's where the difference comes.

Q So there are 20,000 deaths from lung cancer among never smokers in the United States?

A Yes.

Q And you estimate that there are about 137,000 among current and former smokers?

A Yes.

Q If in the 1960's and 70's two-thirds of the population was ever smokers and smoking did not have an effect or an association with lung cancer, you would anticipate a number of cases among the smokers to be -- and former smokers to be twice as high as that among the nonsmokers because of the difference in the size of the populations, is that

correct?

A You mean if it was divided roughly 30,000, 60,000, there was no relationship, yes; under that hypothesis, yes.

Q Now, if eight percent of the lung cancer cases among ever smokers were peripheral cancers and there were 137,000 cases among ever smokers, and there weren't 137,000 in 1970, I believe?

A It would be less than that, sure.

Q How many cases were there in the United States in the 1970's, on an average year in the 1970's?

A If you were sitting in my office I could dig that out for you, but I can't right now.

I would say -- let's say 120,000, maybe, because it was increasing pretty rapidly then.

Q That was among smokers and nonsmokers?

A Yes.

Q If there were 110,000 deaths from lung cancer among smokers per year in the 1970's and eight percent of those cases were peripheral lung cancers, that would mean that there would be 8,800

deaths a year of peripheral lung cancer among smokers during that period, is that correct?

A Roughly that order of magnitude, yes.

Q If during that same time there were 15,000 to 20,000 deaths from lung cancer among nonsmokers, and the most common form of lung cancer among nonsmokers was peripheral carcinoma?

A You have to assume -- you would have to assume under this hypothesis that there were fewer than 15,000 to 20,000 deaths among nonsmokers because the base is only 120,000 rather than 153,000.

Q Well, if thirteen percent of the individuals who had cancer, lung cancer, at that time were nonsmokers and the base, the total base, amount were 120,000, then there would be 15,600 deaths among nonsmokers?

A Something around that order of magnitude.

Q If there were 15,600 deaths from lung cancer among nonsmokers per year during that period of time, and the most common form of cancer, lung cancer, during that time among nonsmokers were peripheral cancers as opposed to central cancers,

one would expect 8,000 or more deaths per year from peripheral cancer among nonsmokers during that period, is that correct?

A We don't know what proportion there is among nonsmokers as opposed to among smokers.

Q Well, among nonsmokers the common location of the cancer was peripheral, is that correct?

A Most common location, yes.

Q So if there were --

A So say if there is 15,000, maybe half of them or so -- all right.

Q So 8,000 or more of the lung cancer deaths among --

A I thought you said there were only 8,000 total?

Q I'm sorry, there was 15,600 total.

A But the peripheral cancers we said was eight percent.

Q That was of the smoking population?

A Of the smoking population, so all right, okay.

Q So, in fact, the number of lung cancer deaths from peripheral carcinoma among nonsmokers,

lifetime nonsmokers, using the calculus that you have established, would have been presumably over 8,000 per year, which is equal to the number of lung cancer deaths among smokers from peripheral carcinoma, is that correct?

A Certainly the percentage -- the percentage of peripheral cancers who never smoked would be higher in -- percent of peripheral adenocarcinomas among nonsmokers would be higher than the percent of peripheral cancers among smokers.

I am not quite sure of the figures you outline, I would have to look at them more carefully.

But if it's -- if you are saying that there is eighty-seven percent versus thirteen percent, that's about what, six times, seven times higher.

I would doubt if the percent of peripheral -- it would mean that the percent of peripheral cancers would have to be about eight percent higher in the never smoked than the smokers.

I doubt if that's true. I don't know, I would have to sit down and figure it out.

MR. SHEFFLER: What was the last thing you said?

THE WITNESS: I said I would doubt if that would be true, but I would have to look at the figures more carefully.

Q You have not done so?

A No, of course not.

MR. GROSSMAN: It's 5:00, this is when we said we would break.

Thank you very much for your time, we will see you in the morning.

THE WITNESS: Okay.

_____

LAWRENCE GARFINKEL

Subscribed and sworn to before me this_____day of_____, 1995.

_____

NOTARY PUBLIC

E X H I B I T S

C E R T I F I C A T E

I, STEPHEN J. MOORE, a Shorthand Reporter and Notary Public of the State of New York, do hereby certify:

That, LAWRENCE GARFINKEL, the witness whose deposition is hereinbefore set forth was duly sworn, and that such deposition is a true record of the testimony given by such witness.

I further certify that I am not related to any of the parties to this action by blood or marriage; and that I am in no way interested in the outcome of this matter.